Asbestos Industry Immunity Bill

A Victim's Perspective on

SB1123 / HB5851

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ASBESTOS INDUSTRY IMMUNITY BILL (SB1123)

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WHAT'S WRONG WITH THE ASBESTOS INDUSTRY IMMUNITY BILL – SB1123 / HB5851

Corporate Attack on Working Families:

- It is estimated that 90% of asbestosis, pleural asbestos scarring and lung cancer victims would be precluded from filing claims against responsible parties!
- The truth about asbestos disease cases is that every asbestos disease victim's case in Michigan Court is already required by law to have a diagnosed active, present lung disease.
- Most asbestos disease case defendants in Michigan are out of state corporations.
- SB1123 / HB5851 may prevent Michigan asbestos victims from filing bankruptcy claims with responsible asbestos product manufacturers.
- SB1123 / HB5851 would shift medical expenses from responsible manufacturers to State funded Medicaid, Medicare, private institutions and the victims of asbestos.

Backlog in Courts Will Be Created!

- There is no asbestos disease case crisis in our State Courts. Asbestos disease cases in Michigan continue to move quickly through the Court system, usually within two years.
- Michigan already has adopted the most severe products liability tort reform in the country including:
  - Caps on jury awards
  - Adoption of several liability
  - Limitations on seller liability, etc.
- SB1123 / HB5851 will create a crisis by preventing the Courts from consolidating similar cases resulting in delay, backlog of cases and waste of judicial resources.
- Additional Trial before Trial will be created.

Medical criteria in SB1123 / HB5851 are NOT based on accepted medical and scientific standards!

Present legal (Wayne County Circuit Court Order) and medical threshold in Michigan to a non-malignant asbestos disease lawsuit, only allows legitimate cases to proceed:

- Current law requires diagnosis of asbestosis based on Michigan medical standard of care – Uses ATS (American Thoracic Society) criteria for the diagnosis of non-malignant asbestos disease:

  (a) Occupational history of asbestos exposure; plus
  (b) Proper latency period; plus
  (c) NIOSH Certified ILO B-reading of chest x-ray positive for asbestosis between 1/0 (early stage) and 3/3 (advanced asbestosis, death imminent) or pathologic diagnosis; plus
  (d) Physical examination and testing; plus
  (e) Written diagnosis and prognosis of asbestos induced disease, as well as a statement of material reviewed, significant medical findings, tests performed, interpretations of physical examination and an opinion on whether or not any abnormality found is related to asbestos exposure by a qualified physician.

New Arbitrary Medical Hoops SB1123 / HB5851 would Create (not based on accepted science and medicine):

New Hoops for Asbestosis and Pleural Asbestos Disease:

1. Arbitrary requirement of ILO rating 2/1.
2. Arbitrary B-2 requirement for asbestos pleural disease.
3. Arbitrary Permanent Impairment of Lung Function.
4. Arbitrary physician hoops and restrictions – limits types of doctors who can testify – requires that doctors must collect insurance payments for the victims care.
5. Only quality 1 x-ray considered – NIOSH ILO system provides for reading quality levels of 1,2,3 or determining x-ray is unreadable.

As to Lung cancer:

Currently Michigan lung cancer cases must meet the medical profession standard of care for diagnosis and asbestos causation based upon accepted medical criteria including ATS and the American Board of Pathologists standards.

New Hoops for Lung Cancer:

1. Arbitrarily requires a diagnosis of non malignant asbestosis before lung cancer can be linked to asbestos.
2. Arbitrary non-medical science based length of exposure hoops. Additionally, SB1123 / HB5851 would have you disregard actual asbestos exposure and arbitrarily only count exposure prior to 1971; if between 1971 and 1979, only count each year for one half year; exclude exposure after 1980 even though asbestos products continued to be sold and exposures continued to occur!
3. Arbitrarily excludes testimony from Board Certified Occupational Medicine Specialists.

Why the Bill is Unconstitutional:

1. Special interest legislation for the benefit of the asbestos industry - violation of equal protection under our laws.
2. Retroactive application. – Termination of vested right.
3. Contingent Fee Contracts Under Michigan Law are abrogated; SB1123 / HB5851 sets a 20% Contingent Fee.
4. SB1123 / HB5851 would legislate a lower arbitrary cap on jury awards for asbestos companies who will be treated differently than any other product manufacturer under Michigan Law.

Conclusion:

All asbestos cases in Michigan already have a legitimate diagnosis of active asbestos disease under accepted medical and scientific standards. If the legislations purpose is to avoid frivolous claims of persons without asbestos disease then we would respectfully suggest that the Wayne County Circuit Court Case Management Order regarding asbestos litigation be adopted. That order already requires a narrative diagnosis of asbestos disease by a licensed physician taking into account the new **American Thoracic Society Criteria for the Diagnosis and Initial Management of Non-Malignant Disease Related to Asbestos (2004) and other accepted medical authority.**

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STATE OF MICHIGAN

IN THE CIRCUIT COURT FOR THE COUNTY OF WAYNE

IN RE: ALL ASBESTOS PERSONAL INJURY CASES

Case No. 03-310422-NP

ORDER NO. 14 (Case Management Order)

At a session of said Court held in the Courthouse, Detroit, Michigan on NOV 2 1 2003

Present: Honorable Robert J. Colombo, Jr.
Circuit Court Judge

The Court having consolidated the personal injury, premises and maritime asbestos cases ("**Asbestos Litigation**") into one docket and having met with representatives of the plaintiffs and defendants, and recognizing the need to reduce the amount and duplication of paper work, time and effort by the parties and the Court in the resolution of the cases, and in order to further promote the interest of justice to all parties, hereby orders the following after providing a Table of Contents for reference:

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Answers

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Defendants' Discovery

I. APPLICATION

A. Applicability of Case Management Order

The Court hereby orders that the Asbestos Litigation shall be governed by this Case Management Order (hereafter "**Order**"). This Order vacates and supersedes all prior discovery orders and Case Management Orders #1-11 and 13 in Wayne County Circuit Court Case No. 93-325280-NP. This Order does not vacate or supercede any applicable Order Scheduling Discovery and Trial Dates, timeout order or Order No. 12, Motions in Limine, in Wayne County Circuit Court Case No. 93-325280-NP.

B. Types of Action

This Order shall apply to all presently pending and future Asbestos Litigation.

C. Applicable Court Rules

Unless otherwise provided by this Order, the current Michigan Court Rules and the current Local Court Rules for the Third Judicial Circuit shall apply. However, Asbestos Litigation shall not be submitted for case evaluation.

D. Electronic Filing System

All cases in the Asbestos Litigation are assigned to the electronic filing and service project known as and hereafter referred to as the "**Electronic Filing System**" as established by an agreement between the Vendor or any successor system and the Wayne County Circuit Court.

II. CASE PROCEDURES

A. Case and Counsel Identification

1. Case Identification

At the time of filing of the complaint in any case subject to this Order, counsel shall identify to the employee of the County Clerk that the case is a personal injury asbestos case to be

filed on the Asbestos Docket. The right hand top corner of the complaint shall be marked "**Asbestos Docket.**"

2. Master Service List

The court shall maintain a current Attorney Roster of all counsel active in cases on the Asbestos Docket and shall circulate that list from time to time among all counsel. In any instance where more than one attorney from a single law firm or company has appeared as counsel, whether or not that firm represents one or more parties, service by the court shall be sufficient for all purposes if served on one of the firm's attorneys, who have appeared. It shall be that attorney's obligation, as an officer of the court, to forward or circulate those Orders, documents, papers or things as to assure delivery to all others in that firm who are affected or interested.

3. Identification of Attorney or Party

It is the duty of each party's attorney, or the party itself if not represented by counsel, to provide the Court with the attorney's or the party's full name, firm or company name, current address, telephone number, fax number and email address so the Court can promptly locate and/or contact the attorney or the party, if necessary. All attorneys and/or parties shall provide the Court with immediate notice of any change of address, telephone number, fax number or email address for any attorney handling Asbestos Litigation.

B. <u>Steering Committee</u>

1. Steering Committee Formation, Purpose and Duties

A steering committee shall be formed for the purpose of meeting with and advising the Court on matters and issues, the resolution of which will promote justice to all parties. The steering committee shall:

a. meet with the Court at least once every six months;

b. discuss with the Court the scheduling of discovery and assignment of trial dates;

c. review the flow of information and petitions for additions to the Court's Master File;

d. suggest resolution of discovery problems;

e. discuss other matters related to discovery and trial as the Court may choose;

f. draft master pleadings and discovery documents; and

g. address all issues relating to the Electronic Filing System.

Membership on the steering committee shall be by appointment of the Court and may be periodically changed by the Court. No order shall be entered based upon discussions that the Court may have with the steering committee without notice to all counsel of record with an opportunity of counsel to discuss in open court their respective views and opinions and to present a legal brief in support of their respective position.

2. The Plaintiffs' Steering Committee Members

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C. Pleadings

1. Master File

The court has created a Master File for the personal injury asbestos cases under the name "In Re: All Asbestos Personal Injury Cases" and Civil Action No. 03-310422-NP ("**Master File**"). Before filing an Order, pleading, motion or other document in the Master File, a party must obtain a number from the Court that will be placed on the front page of the document. The Court and the Vendor shall maintain a Table of Contents for the Master File. All Orders,

pleadings, motions and other documents shall, once filed and docketed in the Master File, be deemed filed and docketed in each individual case to the extent possible. The Wayne County Clerk and the Vendor shall each maintain the Master File.

2. Caption; Separate Filing

Orders, pleadings, motions and other documents shall be filed in the Master File if they apply to all personal injury asbestos cases and the approval of the Court has been obtained. Orders, pleadings, motions and other documents only applying to a particular case[s] shall indicate the case[s], name and the civil action number[s] for that case[s] and shall be filed only in the court file for that case[s].

3. Master Order Numbers

All Orders which by their nature are to be filed in the Master Pleading File shall be numbered consecutively and shall bear the legend "Order No. ___." The courtroom clerk will assign the appropriate number when the order is entered by the court and will index the Order.

4. Complaint

Plaintiffs' standard complaints have been filed in the Master File, (#11 Plaintiffs' Standard Complaint Living Plaintiff and Spouse; #12 Plaintiffs' Standard Complaint Deceased Plaintiff; #13 Plaintiffs' Standard Complaint Living Plaintiff and Spouse With Premises Count; and, #14 Plaintiffs' Standard Complaint Deceased Plaintiff With Premises Count).

a. A case may be commenced by filing and serving a Notice of Complaint or a Notice of Complaint in a Wrongful Death Case, attached as Exhibits A and B and designated by number which standard complaint the suit is based on.

b. Plaintiffs shall supply a copy of the standard complaint or any standard pleadings upon written request by an adverse party.

c. Plaintiffs shall not file consolidated personal injury asbestos cases joining more than one plaintiff in the same case. This provision does not preclude joining a loss of consortium claim.

d. With respect to all Asbestos Litigation filed on or after March 28, 1996 (the effective date of MCLA 600.2957), Plaintiffs shall include in their short form Complaints:

i. the identity of all then known non-parties whose asbestos containing products plaintiff has reason to believe he or she was or may have been exposed to during his or her work career;

ii. then known years of exposure to asbestos;

iii. known job or exposure sites; and

iv. dates on job or exposure sites, if known and subject to plaintiffs' right to update after investigation of the case and review of the Social Security printout and in accordance with this Order's deadline for submission of a final product I.D. Brochure.

e. When filing a short form complaint in a case where an allegedly injured plaintiff has died, plaintiff's counsel shall attach, where available, a copy of the Letters of Authority and death certificate. If not filed with the short form complaint, the Letters of Authority and death certificate shall be served on all defendants once available to plaintiff's counsel.

f. When filing a short form complaint, plaintiff's counsel shall not disclose the plaintiff's social security number or date of birth in the complaint or in any attachment to the complaint. In order to avoid the public disclosure of private information, plaintiff's counsel shall serve Defendant, with the plaintiff's social security number and date of birth with service of the short form complaint in a separate document which shall not be filed with the Court.

5. Answer

Defendants' standard answers to Plaintiffs' standard complaints have been filed in the Master File, (#21 Defendants' Standard Answer to Plaintiffs' Master Complaint Living Plaintiff and Spouse; #22 Defendants' Standard Answer to Plaintiffs' Master Complaint Deceased Plaintiff; #23 Defendants' Standard Answer to Plaintiffs' Master Complaint Living Plaintiff and

Spouse With Premises Count; and, #24 Defendants' Standard Answer to Plaintiffs' Master Complaint Deceased Plaintiff With Premises Count).

a. Each defendant shall answer by filing in writing the number of the designated standard answer it is pleading.

b. Defendants will supply a copy of the standard answer or any standard pleadings upon written request by an adverse party.

c. If affirmative defenses are raised which are not part of the affirmative defenses in the master file, said new affirmative defenses shall be considered filed in a case by including same specifically in writing in the first responsive pleading

d. Filing of an appearance on behalf of a defendant does not waive any challenge to jurisdiction nor shall it be deemed to be consent to jurisdiction.

6. Reply to Affirmative Defenses

It shall not be necessary for a plaintiff to reply to affirmative defenses and all affirmative defenses shall be deemed to be denied.

7. Answer to Third Party or Cross Complaints

It shall not be necessary for third party or cross-defendants to answer third party or cross-complaints and all allegations of a third party or cross-complaint shall be deemed denied by the third party or cross-defendants unless a third party or cross-defendant files a response indicating otherwise.

8. Maritime Asbestos Cases

Complaint:

a. Plaintiffs in maritime asbestos cases shall file a complaint alleging any and all appropriate causes of action and shall not file Plaintiffs' Standard Complaints set forth above.

b. Plaintiff shall file Initial Data Form in conformity with Exhibit C attached to this Order with every maritime asbestos complaint.

Answer:

a. Each defendant shall file an Answer specific to each Plaintiff's complaint and shall not file the Standard Answer set forth in paragraph C5, above.

9. Amendment of Pleadings to Add Parties

In the event that a plaintiff determines there are additional parties that should be added to a pending action, plaintiff shall obtain an ex-parte order permitting said amendment to add parties. The order shall provide the date that the summons is to expire, which shall be 91 days from the date the summons is issued. Once the order adding parties has been entered by the Court, the plaintiff's counsel obtaining that order shall immediately serve notice that a specific party has been added to a case on the defense counsel who currently represents the newly added defendant(s) or, if unknown, on the counsel listed on the Attorney Roster. In addition, plaintiffs' counsel shall serve the amended summons and complaint on the newly added defendant(s) at least thirty-five (35) days before the final Brochure is due. No other parties shall be added to the action, except upon motion to the court for good cause shown.

10. Notice of Fault of Non-Parties

Within 91 days from the service of the Complaint, defendants shall file a notice to plaintiffs of their intent to assert that a non-party is wholly or partially at fault by complying with the requirements of MCR 2.112(k).

D. <u>Master Interrogatories And Answers To Interrogatories</u>

1. Plaintiffs' Interrogatories

a. Plaintiffs shall file in the Master File two joint sets of master interrogatories and request for production of documents to defendants (#31 Plaintiffs' Standard Interrogatories to Defendant and Request for Production of Documents for Non-Premises Liability Defendants and #32 Plaintiffs' Standard Interrogatories to Defendant and Request for Production of Documents for Premises Liability Defendants).

b. A defendant shall serve answers to interrogatories on each plaintiff attorney one time. The answers to interrogatories shall not be filed in the Master File. Thereafter, each defendant shall be obligated to update the answers as additional information becomes known to defendant.

2. Defendants' Interrogatories

a. Defendants shall file two master sets of interrogatories covering liability and damage issues, (#41 Defendants' Third Master Interrogatories and Request for Production of Documents to Plaintiff/Decedent and #42 Defendants' Master Liability Interrogatories and Request for Production of Documents to Plaintiff(s)).

b. Each plaintiff's attorney need only serve one set of answers to master set regarding liability on each defendant. Thereafter, plaintiffs shall update answers as new information becomes known.

c. Each plaintiff shall serve answers on each defendant regarding plaintiff's personal data interrogatories.

3. Maritime Interrogatories and Notice and Request For Production of Documents

a. Plaintiffs' Interrogatories

(i) Plaintiff shall file in the Master File, Plaintiffs' Supplemental Maritime Interrogatories and Notice and Request for Production of Documents to Defendant (#51 Plaintiffs' Standard Maritime Interrogatories to Defendants and Request for Production of Documents – Non Jones Act Defendants).

(ii) A defendant shall serve Answers to Interrogatories on each plaintiff attorney one time. The Answers to Interrogatories shall not be filed in the Master File. Thereafter, each defendant shall be obligated to update the Answers as additional information becomes known to defendant.

b. Defendants' Interrogatories

Defendants shall file in the Master File, Defendants' Standard Maritime Interrogatories and Request for Production of Documents for Living Plaintiff (#52 Defendants' Standard

Maritime Interrogatories and Request for Production of Documents for Living Plaintiff (Master Supplemental Interrogatories to GAF Pattern)).

(i) Each plaintiffs' attorney need only serve one set of answers to master set regarding liability on each defendant. Thereafter, plaintiff shall update answers as new information becomes known.

(ii) Each plaintiff shall serve answers on each defendant regarding plaintiff's personal data interrogatories.

4. Supplemental Interrogatories

Supplemental Interrogatories may be served by any party as to matters and issues not covered by the Master sets of Interrogatories.

E. Brochure

1. Elements of Brochure

The elements of the Brochure are intended to include reasonable notice to defendants of product identification information reasonably available to plaintiffs and/or their counsel. The information contained in the Brochure shall be sworn to by the plaintiff's attorney as true and accurate information prior to the plaintiff's deposition or before the date of trial, whichever is earliest. To the extent reasonably available, the elements shall include:

a. the specific product name, or a description of the product if the specific product name is unknown, and the manufacturer, supplier, distributor, seller of such product and contractor using such product;

b. name of employers, if applicable;

c. specific location of job or exposure site where plaintiff worked or was exposed and where products were used or observed, including the name and address of the job or exposure site;

d. the dates plaintiff either worked at a job or exposure site or was exposed to products containing asbestos;

e. the identification of all other products which were used on the same job or exposure site and at the same time which may contain asbestos;

f. the names, addresses and telephone numbers of person(s) who can provide product identification or exposure testimony for plaintiff at each job or exposure site;

g. the identity (by document number or otherwise) of any writing supporting product identification; and

h. a brief synopsis of other evidence which plaintiff claims establish product identification against a defendant, whether or not directed to a specific job or exposure site.

2. Evidence Excluded if not in Brochure

Except for good cause shown, a plaintiff shall be prohibited from introducing product identification evidence where it has not been disclosed in the Brochure.

a. Amendment of Brochure

If a defendant conducts a deposition of a co-worker and during the deposition additional exposures or job sites are developed by a defendant, plaintiff may move to amend the Brochure to add the additional exposures or job sites developed by submitting a proposed order to amend stating specifically the information plaintiff requests be added to the Brochure under the seven (7) day rule provision of MCR 2.602(B)(3) and a defendant may file objections with the court by brief or oral argument demonstrating to the court that good cause exists to not allow the amendment.

b. Information Divulged at Co-Worker Deposition Not in Brochure

If a defendant is not present at the deposition of the co-worker because the Brochure did not include evidence that the co-worker would identify defendant's products or defendant's job site, then any information developed during the deposition regarding the said defendant cannot

be used against the defendant at trial unless another party of record obtains an order from the court based upon a showing of good cause. This shall not be applicable to a co-worker's deposition taken before the Brochure is filed.

c. Lack of Product Identification at Deposition of Fact Witnesses

Whenever a defendant attends the deposition of a fact witness listed in plaintiff's Brochure, because the Brochure indicated that witness would testify plaintiff was exposed to the defendant's asbestos containing product(s) or worked at a premises liability defendant's job or exposure site, and the witness during the course of the deposition is asked if he can identify that defendant's product(s) or job or exposure site, or exposure to plaintiff, as identified in plaintiffs' Brochure, and the witness states under oath that he cannot identify said defendant's product(s) or job or exposure site as specified in plaintiffs' Brochure and at a time when plaintiff reasonably may have been exposed to that product or job or exposure site, the defendant may file a motion requesting costs for the time incurred in the preparation, travel to, and attendance at said deposition. Unless plaintiff is able to demonstrate to the court that a reasonable basis existed at the time of filing of the Brochure upon which to believe the witness would identify the defendant's product(s) or job or exposure site and exposure to plaintiff, the court shall assess costs in an amount which the court deems to be reasonable and just under the circumstance. This provision shall not be applicable to a co-worker's deposition taken before the Brochure is filed.

d. Witnesses

The Brochure shall identify which witnesses plaintiffs reasonably believe can identify a defendant's product(s) even if unable to place the product(s) on a job or exposure site at a particular time, if plaintiffs reasonably believe the witnesses will be able to testify that more

likely than not a plaintiff was exposed to that defendant's product(s) during plaintiff's working career.

e. Exposure Disclosure

In plaintiffs' answers to interrogatories and Brochure, plaintiffs shall disclose information concerning all exposures to asbestos-containing products known or reasonably available to plaintiffs or plaintiffs' counsel, including exposures to products manufactured and/or distributed by companies which are not defendants to the action. The above shall not require plaintiffs or plaintiffs' counsel to search for all non-defendant asbestos related exposure.

f. Admissibility

While plaintiffs' attorney may sign the Brochure, the Brochures shall be treated as responses to interrogatories and shall be admissible as evidence.

F. <u>Discovery Schedule</u>

1. Due Date for Answers to Interrogatories

The Interrogatories in the Court's Master File must be answered one hundred eight-two (182) days after the date the complaint was filed. If a defendant is added to a case by an amendment of pleadings to add parties, the defendant shall answer the Master Interrogatories within sixty-three (63) days of the date said defendant was served with the amended complaint or within one hundred and eight-two (182) days of the filing of the original complaint, whichever is later. All plaintiffs and defendants must serve Answers to the Master Interrogatories pursuant to said deadlines unless a party has obtained written consent from opposing counsel that it is not necessary to do so or on Order of the Court.

2. Due Date for Brochure and Proposed Deposition Schedule for Plaintiffs and Personal Representatives

Plaintiffs' attorney must serve the designated defense counsel with a proposed deposition schedule for plaintiffs and/or the Personal Representatives, two weeks before the Brochure is due. Plaintiffs' Brochure shall be served on the date set forth in the schedule attached to the Order Scheduling Discovery and Trial Dates. (Approximately six (6) months before the trial date).

3. Medical Authorizations

A plaintiff shall provide medical authorizations to any party who advises plaintiff that he/she will be medical counsel for the case, whether it be a medical counsel on behalf of a group of defendants or a single defendant.

4. Order Scheduling Discovery and Trial Date

The Court shall enter and serve in February and August of each year, an Order Scheduling Discovery and Trial Dates attaching a schedule as shown in Exhibit D.

a. Plaintiff's Deposition

(i) Any party may take the deposition of the plaintiff at any time after the filing of the Brochure and in accordance with the deadlines set by the Order Scheduling Discovery and Trial Dates for the case, absent special circumstances or meriting a deposition of the plaintiff at an earlier date.

(ii) Within 28 days from the date established for the completion of plaintiffs' depositions, defendants shall file a Brochure as to any non-party identified as a result of information obtained from plaintiffs' Brochures or at plaintiffs' deposition. Defendants' Brochures shall comply with the Brochure elements set forth in this Order. Each defendant may

rely upon any other Brochure filed by any other defendant. A defendant not filing a Brochure is not required to file a reliance; a reliance is deemed filed by each defendant.

(iii) Unless otherwise ordered by the Court or stipulated to by all parties, before a plaintiff's de bene esse deposition is taken, defendants shall have reasonable opportunity to obtain a discovery deposition, if same was not previously taken.

(iv) Prior to the discovery and the de bene esse deposition of the plaintiff, the plaintiff shall provide all defendants with answers to the standard set of interrogatories, a Brochure, any and all available medical records of the plaintiff and an affidavit which sets forth adequate reasons why the plaintiff's testimony must be preserved by a de bene esse deposition.

b. Non-Medical Fact Witness Depositions

Non-medical fact witness deposition cut-off dates and trial dates shall be in accordance with the deadlines set in the Order Scheduling Discovery and Trial Dates (see Exhibit D) for the case.

c. Witness Lists

All parties shall serve adverse parties with witness lists in accordance with the deadlines set by the Order Scheduling Discovery and Trial Dates for the case; however, plaintiffs and defendants may serve, but not e-file, a master witness list, which shall apply to all pending and hereinafter filed asbestos bodily injury cases. The serving of the master witness list by plaintiffs and defendants shall satisfy the requirements of exchanging a witness list pursuant to the order scheduling discovery and trial dates for each individual case. If a plaintiff or a defendant chooses to supplement its witness list in an individual case, it shall be done in compliance with the Order Scheduling Discovery and Trial Dates for that case.

d. Exhibit Lists

Exhibit lists shall not be filed by any party unless ordered by the court.

e. Medicals

(i) Except for good cause, plaintiffs shall provide the defense medical counsel assigned to the case with all tissue, slides and x-rays with notice to other defense medical counsel in accordance with the schedule attached to the Order Scheduling Discovery and Trial Dates (approximately six (6) months prior to the trial date).

(1) The defense medical counsel assigned to that case shall coordinate activities for purposes of sharing the material and to the extent possible the cost of review of said material by defense expert(s).

(2) In the event cooperation cannot be achieved among medical counsel for defendants in this regard, the Court will meet informally, in chambers, with the counsel involved, and assign the limits for sharing the material.

(ii) Plaintiffs shall serve all attorneys of record with a copy of plaintiffs' expert's medical report(s) which shall include an opinion on diagnosis and prognosis, as well as a statement of material reviewed, significant medical findings, tests performed, results of test(s), interpretations of tests, interpretations of each physical exam, if performed, and an opinion on whether or not any abnormality found is related to asbestos exposure. The letter shall not contain an opinion regarding issues of liability. Plaintiffs shall serve the report(s) in accordance with the deadlines set by the Order Scheduling Discovery and Trial Dates. (Exhibit D).

(iii) Defendants shall return to plaintiffs' attorney any and all items provided to defendants' attorney, pursuant to paragraph 4 in accordance with the deadlines set by the Order Scheduling Discovery and Trial Dates for the case.

(iv) Defendants shall serve plaintiff with a medical expert's(s) report(s) which will set forth the same type of information as plaintiff is required to provide in plaintiffs' expert's(s)

report(s). Each report is to be served on the adverse party in accordance with the deadlines set by the Order Scheduling Discovery and Trial Dates for the case.

(v) In the event plaintiff intends to provide medical rebuttal evidence at the time of trial, plaintiff must serve the defendants with a medical expert's(s) report(s) which will include additional and revised opinions of plaintiffs' expert(s). Plaintiffs' rebuttal report(s) shall be served on all defendants of record on the date the matter is set for trial.

(vi) A party relying on an economist need only serve one detailed report of opinions and theories and/or the economist shall submit to one deposition. Thereafter, the party retaining the economist need only serve an opinion regarding the amount of losses claimed by each plaintiff. If new theories are developed, a new report must be served on the adverse party and/or the economist shall be produced for another deposition.

(vii) Discovery depositions of expert witnesses may be taken at any time except during trial hours.

f. De Bene Esse Depositions

De bene esse depositions of fact witnesses may be taken up to the date set for trial. De bene esse depositions of expert witnesses may be taken at any time except during trial hours.

G. <u>Motions</u>

1. Motion Days

Judge Robert J. Colombo shall hear all motions on the Asbestos Litigation on every third Friday of the month at 8:30 a.m., except that the following motions may be heard on any Friday that Court is in session:

a. to approve settlement;

b. to approve distribution of proceeds in a wrongful death action; and/or

c. to enforce compliance with the terms of this Order relating to conduct occurring after a case has been up for settlement conference/trial.

No dispositive motion may be filed within seven (7) days of commencement of or during a "time out" period.

2. Emergency Motions

If an emergency situation exists, a party wishing to file an emergency motion shall obtain a date from the court and shall advise all appropriate parties of record of the date and time of the motion hearing.

3. Non-Dispositive Motions

a. Whenever a non-dispositive motion is directed to one defendant or made by one defendant, it shall only be necessary to serve copies and responses upon counsel for the opposing party for that specific motion.

b. All counsel, however, shall be served with a cover letter summarizing the nature of the motion, the relief sought and the date set for hearing.

c. Upon request, copies of such pleadings and documents filed with the court shall be served on other counsel in the case.

4. Dispositive Motions

a. Any defendant may serve a dispositive motion based on a lack of product identification at any time after the service of the Brochure, or in the case of the premises defendants, at any time after plaintiffs have answered the master set of interrogatories.

b. For good cause, a defendant, who contends that there is little likelihood of product identification, may serve a dispositive motion based upon lack of product identification at any time after the filing of the answers to interrogatories. If any defendant believes special circumstances exist which merit a dispositive motion prior to plaintiff serving answers to interrogatories, that party may file a motion pursuant to the requirements set forth within this order.

c. All dispositive motions and responses must be served on all parties of record.

5. Motions Applicable to More Than One Case

When a motion applies to more than one case, the following procedure shall be followed:

a. The praecipe, the motion and all other related documents shall have the caption and the number of the first case filed (oldest case) to which the motion applies.

b. The praecipe and motion shall indicate under the case number, "Applies to all listed cases attached to motion."

c. Attached to the motion shall be a list of all cases to which the motion applies. The list shall be compiled by attaching the court sticker for each case. The cases shall be listed numerically beginning with the first case filed (oldest case) to which the motion applies.

d. A motion fee shall be paid for each case.

e. The motion shall only be filed in the case file of the first case filed (oldest case) to which the motion applies. The motion shall not be filed in any other case file.

f. The same procedure applies to an answer to a motion, except that attached to the answer shall be a list of all cases to which the answer applies.

g. An Order shall be entered and filed in every case file to which the motion applies. The Order shall indicate **"Relief is granted pursuant to the motion filed in (insert case number of the first case filed (oldest case) to which the motion applies.)"**

6. Motions Applicable to More Than One Party

Motions seeking relief which would be applicable equally to all parties in the same position or situation as the movant shall be filed by one moving party. All other parties, who are equally affected by such motion, shall refrain from filing concurring motions which repeat any argument or position already made by the original movant. Relief may be granted or denied as to all parties in the identical legal position of the movant without the necessity for other parties to file a motion for relief. A party who does not wish to be affected by the motion may opt out by letter to the court.

7. Motions Indicating Application to Parties

Motions which apply to more than the movant shall have endorsed on the praecipe and on the first page of the motion the legend, "This Motion Applies to All (e.g., Defendants)," or "This Motion Applies Only to (e.g., Defendants Smith, Jones and Doe)."

8. Motion Titles

All motions shall be titled, specifically indicating briefly the nature of the relief sought, e.g., "Defendant's Motion for Summary Disposition Based On Statute of Limitations," or, "Defendant's Motion for Summary Disposition Based On Lack Of Product Identification."

9. Orders Indicating Application to Parties

All orders entered shall contain a statement on the face of the order, indicating that this order applies to all parties or only to specified parties.

10. Entry of Orders on Unopposed Motions

Motions which are not expected to be opposed may be accompanied by an order noticed under the seven (7) day rule, pursuant to MCR 2.602(B)(3). If no objections are filed within the seven (7) day period, the court will rule on the motion without oral argument. If objections are received within the seven (7) day period, the motion will be heard on the next regular asbestos motion date.

11. Motions for Change of Venue

a. All motions for change of venue shall be made on the form attached as Exhibit E to this Order.

b. The defendants shall have the right to file a motion to decline jurisdiction or change venue but not before the filing of plaintiffs' Brochure and not later than thirty-five (35) days after the filing of plaintiffs' Brochure.

c. Either party may serve interrogatories on opposing parties as to any matter related to the issue of jurisdiction or forum non conveniens.

d. These change of venue provisions shall not apply to maritime asbestos cases.

12. Conference Calls

The Court is available for a conference call in the event of an emergency or unusual circumstances. The moving party shall have the responsibility to bear the costs of the conference call and to make all arrangements to ensure that all parties, who wish to participate in the conference, are made a party to the call.

H. Defense Medical Counsel

1. Identity of Medical Counsel

Defense medical counsel shall advise plaintiffs' attorney assigned to the case of the counsel who will be responsible for the medical development for that defendant(s). The parties recognize that there may be a counsel representing defendants participating in the medical program and that some defendants will be responsible for their own medical.

2. Coordination of Medicals

Defense medical counsel shall coordinate with each other to share x-rays, tissue slides or blocks and obtaining an independent examination of plaintiff. These materials shall be provided to the defense medical attorney assigned to the case.

I. Social Security Printout

1. Application for Social Security Printout

Counsel for plaintiff shall promptly apply for a Social Security Printout at the time of commencement of the lawsuit.

2. Service of Social Security Printout

When the Social Security Printout is received, counsel for plaintiff shall promptly serve each counsel for the defendants with a copy of the Social Security Printout.

J. Theories Of Liability Re: Non-Premises Defendants

Plaintiffs' theories of liability as to the non-premises defendants shall be limited to failure to warn, failure to test, negligent design, breach of implied warranty and gross negligence as to all defendants and exemplary damage claims only as to Defendants Owens Corning Fiberglass, Pittsburgh Corning and Rapid American, to which these Defendants object. All other theories shall be struck from the complaint ninety-one (91) days before the date the matter is set for trial unless there exists a change in the law or factual development regarding any theory struck prior to trial. To prevent a theory from being automatically struck ninety-one (91) days before trial, plaintiffs shall file at least ninety-one (91) days before trial, a motion with the court and demonstrate a change in the law or a new factual development which justifies retention of the theory. This paragraph does not apply to maritime asbestos cases.

K. Damage Claims

All monetary damages claimed, except compensatory damages as to all defendants and exemplary damage claims only as to Defendants Owens Corning Fiberglas, Pittsburgh Corning and Rapid American, to which these Defendants object, shall be struck from the complaint ninety-one (91) days before the date set for trial unless there exists a change in the law or factual development regarding the damage claim struck prior to trial. To prevent a damage claim from being automatically struck ninety-one (91) days before trial, plaintiffs shall file at least ninety-one (91) days before trial, a motion with the court and demonstrate a change in the law or a new factual development which justifies retention of the theory. This paragraph does not apply to maritime asbestos cases.

L. Autopsy

1. Autopsy Prior to Burial or Cremation

In the event of death of a plaintiff and an autopsy is done prior to burial or cremation, defendants are not entitled to have notice of the autopsy or have a representative present during the autopsy.

2. Right to Analyze Specimens, Blocks or Slides

Defendants are entitled, however, to have an expert(s) of their choice analyze whatever tissue specimens, blocks or slides that were created during the autopsy.

M. Exhumation

1. Notice of Exhumation

If exhumation, with or without autopsy is scheduled, attorney for the plaintiff shall give prompt and reasonable notice of date, time and place of the exhumation and autopsy, if one is to performed, to the defense medical counsel, and any other attorney doing a medical exam for his client, so that said counsel may arrange to have a physician of their choice present at the exhumation, tissue removal, and/or autopsy, solely for the purpose of observation. Said physician shall have no right or opportunity to disturb the corpse or interfere in any manner with the investigation being performed by the pathologist conducting the autopsy.

2. Limited Examination by Defense Expert of Plaintiff Scheduled Exhumation

Upon motion for good cause shown, the court may allow the physician selected by the defendants to conduct a limited examination of the corpse and/or removal of tissue, if exhumation by the plaintiff has been scheduled.

3. Court Order Required for Exhumation

Nothing in this action shall be interpreted to provide a right to the defendants for exhumation without prior order of the court.

N. Submission Of Releases And Settlement Payments

1. Releases

Unless otherwise agreed between the parties, a settling defendant(s) shall serve Releases to plaintiffs no later than fourteen (14) days after a Settlement Agreement is reached. In the event a defendant fails to serve Releases consistent with the Order, interest may accrue from the date the settlement was reached in the event of late payment of the settlement proceeds.

2. Settlement Payments

Unless otherwise agreed between the parties, payment of all settlement proceeds by a specific defendant for a particular trial group shall not be due until twenty-eight (28) days after a specific defendant has received all properly executed Releases and all Orders of Consent Judgment or all Orders for Authority to enter into a settlement for all cases in the particular trial group relating to a specific defendant. In the event a defendant fails to submit the settlement proceeds consistent with this Order in death cases, interest shall accrue on the settlement proceeds from the date the defendant received the properly executed Release and the Order of Consent Judgment or the Order for Authority to enter into settlement, whichever occurred last. In all other cases, where the defendant fails to submit the settlement proceeds consistent with this Order, interest shall accrue from the date the defendant received the properly executed Release.

a. Death of the Plaintiff

If a living plaintiff becomes deceased within 63 days before the scheduled trial date, or after settlement is agreed upon, then plaintiff's counsel may seek an order from the Court

severing the case from a particular settlement group, and requiring that the defendant comply with paragraphs 1 and/or 2 above with respect to the remainder of that particular settlement group.

b. Other Exceptions

The order described in paragraph II. N(2)(a) above may also be sought if plaintiff's counsel demonstrates that one or more plaintiffs cannot or will not comply with the release signing or Consent Judgment requirements in a reasonably timely fashion.

O. Orders Of Dismissal And Administrative Closing

This Court will accept counsel prepared Order of Dismissals for entry until the Friday before a scheduled trial date. After this date, the Court will require counsel to execute Court prepared stipulations and orders of dismissal and/or administrative closings. In death cases, only, the Court will not prepare a Stipulation and Order of Dismissal, since Consent Judgments will be prepared by plaintiff's counsel. However, in death cases where a defendant is dismissed after the Friday before the trial date, that defendant may serve a stipulation and order of dismissal for entry.

P. Admission Of Attorneys

Attorneys admitted *pro hac vice* may be permitted to engage in the trial of a specific case on the Asbestos docket in accordance with State Bar Rules of Michigan, Rule 15, Section 2.

1. Motion by Active Member of State Bar

An active member of the State Bar of Michigan ("**State Bar**") who appears of record in the case shall file a motion for temporary permission for a foreign attorney to engage in the trial of a specific case.

2. Affidavit of Foreign Attorney

A foreign attorney by Affidavit shall disclose to the court whether or not he/she has been censured, denied admission, or in any way disciplined in any court indicating the full title and address of the action involving the presiding judge (the fact that another judge ordered said file sealed in any respect shall not relieve the attorney of this responsibility for full disclosure).

3. Discretionary Admission

Admission of an attorney *pro hac vice* shall remain totally within the discretion of the Court and the court shall retain full authority to withdraw this privilege.

4. Submission of Motion

Motions for admission *pro hac vice* may be served with an order under the procedure set forth in II. G.

III. ELECTRONIC FILING SYSTEM

A. Application

All cases in the Asbestos Litigation are assigned to the electronic filing and service project known as and hereinafter referred to as "**Electronic Filing System**" as established by agreement between the Vendor or any successor system and the Wayne County Circuit Court.

B. Commencement Date

The Commencement Date for the implementation of the Electronic Filing System shall be determined by the Court. Parties should complete the Vendor's subscriber process no later than December 19, 2003 in order to file, serve and receive service electronically on the Commencement Date. See Exhibit F for the Vendor's contact information.

C. **New Defendant**

If a new defendant is brought into the Asbestos Litigation after the signing of this Order, then counsel for the party bringing in the new defendant shall immediately serve a copy of this Order on counsel for the newly added defendant and shall advise counsel for such newly added defendant of its obligation to participate in the Electronic Filing System. A newly added defendant shall have sixty three (63) days from the date of service of a copy of this Order to become a User in the Electronic Filing System. During the period prior to subscribing to Vendor's Electronic Filing System, that defendant must: 1) bring a 3 ½ inch diskette containing the documents to be filed to the Wayne County Clerk's office to be uploaded to the Electronic Filing System; and 2) serve said documents on plaintiff's counsel in the conventional manner in accordance with the Michigan Court Rules.

D. **Non Parties and Parties Appearing In Pro Per**

While non-parties and parties appearing in pro per are not required to execute a subscription agreement with the Vendor or to become a User, they must file and/or serve all documents in accordance with the Michigan Court Rules. Where a non-party or party appearing in pro per appears in an action and does not become a User, the other parties shall file and/or serve all documents electronically; however, all documents which must be filed and/or served on the non-party or party appearing in pro per must be served on the non-party or party appearing in pro per pursuant to the Michigan Court Rules.

E. **Definitions**

The following terms in this Order shall be defined as follows:

1. **Acknowledgment of Receipt** refers to the online acknowledgement the Vendor provides to a User immediately upon completion of the transmission of Electronic Documents or

Electronic Images to the Vendor's Electronic Filing System. The Acknowledgment of Receipt indicates whether the Electronic Document or Electronic Image is to be filed and/or served and, if served, on whom. The Acknowledgment of Receipt is not a confirmation that the Electronic Document and/or Electronic Image have been accepted for filing by the Office of the Wayne County Clerk.

2. **Confirmation** refers to the email notice the Vendor provides to a User who has transmitted an Electronic Document and/or Electronic Image to the Vendor's Electronic Filing System for filing with the Office of the Wayne County Clerk. The Confirmation notifies the User that the Office of the Wayne County Clerk has received the document for filing. The Confirmation is not a confirmation that the Electronic Document and/or Electronic Image has been accepted for filing by the Office of the Wayne County Clerk.

3. **Electronic Document** means an electronic file of a word processing document that contains almost exclusively text.

4. **Electronic Filing** means the electronic transmission of an original pleading, paper, order, or document to or from the Wayne County Circuit Court via the Vendor's Electronic Filing System. It does not include:

a. a facsimile transmission;

b. a Complaint;

c. an Amended Complaint that adds a party or parties;

d. a Third Party Complaint; or

e. a document filed under seal.

5. **Electronic Image** means an electronic file of a document that has been scanned or converted to a graphical or image format.

6. **Electronic Service** means the electronic transmission of an original document to all other designated recipients via the Vendor's Electronic Filing System. Immediately upon completion of any transmission to the Vendor's Electronic Filing System, the Vendor shall provide the sender with an online Acknowledgement of Receipt of the transmission by the Vendor's Electronic Filing System.

7. **Notification** refers to the email notice the Vendor provides to all parties who are Users in a particular case that one of the parties has electronically filed and/or served an Electronic Document or Electronic Image in that particular case. Upon receipt of the Notification, the User may access the Vendor's Electronic Filing System to review and/or obtain a copy of the Electronic Document or Electronic Image.

8. **Typographical Signature** means the signature on an Electronic Filing that is not in the personal hand of the signing party or a facsimile of a hand signature. Rather, the signing party may type his or her name in the place on the document that would otherwise be hand signed if filed conventionally. The Typographical Signature shall be accompanied by the symbol "/s/" and treated as a personal signature for all purposes under the Michigan Court Rules, inclusive of MCR 2.114.

9. **User** means a party, non-party, or attorney who has executed a subscription agreement with the Vendor. All attorneys serving or filing Electronic Documents and Electronic Images must be licensed to practice law in the State of Michigan.

10. **Vendor** means a private sector firm or other business entity authorized by the Court to provide an Electronic Filing System. A Vendor is contractually obligated to provide specified electronic services to the Bar, the public, and the Court, to transfer electronic filings to

and from the Court, and to provide Notification to all Users served with the Electronic Document or Electronic Image.

The Vendor is identified in Exhibit F.

F. Operation Of Electronic Filing And Service Procedure

1. Obtaining Access to System

Counsel for all potential Users shall promptly take the steps necessary to enable them to electronically file, serve, receive, review and retrieve copies of all pleadings, papers, orders and other documents filed in the Asbestos Litigation electronically by registering with and entering into a subscription agreement with the Vendor.

At completion of the subscription process, the Vendor shall assign a confidential user name and password to the potential User that may thereafter be used by such potential User to obtain access to the Electronic Filing System. This user name and password will permit the potential User to file, serve, receive, review and retrieve electronically filed pleadings, papers and other documents filed in a case; and

No attorney shall knowingly authorize or permit his or her user name and password to be utilized by anyone else. Attorneys may authorize other attorneys or employees of the attorney's law firm and designated co-counsel (where the Vendor has been notified in writing that designated co-counsel may file documents on behalf of the assigning counsel), to receive their own user name and password to file on their behalf. No person shall knowingly use another's user name and password or cause or permit another person to use them without the express permission from the holder of the user name and password.

2. Electronic Filing and Electronic Service

a. Pleadings, Motions, Briefs and Other Documents

Except as provided in Section III.E(4) of the Order, all pleadings, motions, briefs, memorandums of law or other documents required to be filed with the Court in connection with the Asbestos Litigation, and any attachments thereto, shall be filed and served electronically.

b. Discovery and Discovery Responses

Discovery requests and discovery responses (including discovery Brochures) shall not be filed. The parties shall exchange discovery and discovery responses by serving the Electronic Document or Electronic Image electronically. Discovery requests and responses which are served electronically will not be made a part of the Court file. Rather, such discovery shall be available on the Electronic Filing System only to the parties in that particular case.

3. Orders, Opinions and Communications of Court

All judicial rulings, opinions, orders and other communications of Court shall be electronically filed. Such documents shall be signed with a Typographical Signature.

4. Notice of Electronic Filing

The Vendor shall provide Notification to all Users served electronically with an Electronic Document or Electronic Image.

5. Filing Related Documents

All documents relating to a single pleading or paper shall be filed electronically as a single document. For example, a motion, a memorandum in support of the motion, praecipe, notice of hearing, exhibits and related affidavits shall be filed as a single transaction.

6. Electronic Service of Pleadings and Other Documents

All Users shall serve other Users electronically through the Electronic Filing System. Users shall receive all documents electronically filed and served upon them via access to the Vendor's Electronic Filing System. The Electronic Service of a pleading or other document in the Electronic Filing System is considered valid and effective service on all served Users and shall have the same legal effect as conventional service of an original paper or document. A User is not required to conventionally serve a paper copy of the electronically filed document on the other parties in the case.

7. Proof of Service Not Required

Proofs of Service shall no longer be filed with the Wayne County Clerk. The Notification will serve as a substitute for such Proofs of Service.

8. Return of Service

Plaintiffs and Third-Party Plaintiffs shall electronically file returns of service in the Electronic Filing System as Electronic Images.

9. Conventional Filing of Documents

Notwithstanding the provisions of Paragraph III.C(2) of this Order, the following types of pleadings shall be filed according to MCR 2.107:

a. Complaint;

b. Amended Complaint that adds a party or parties; and

c. Third Party Complaint.

Notwithstanding the provisions of Paragraph III.C(2) of this Order, the following types of papers or documents shall be filed conventionally:

a. Sealed Documents:

Although a motion to file documents under seal shall be filed and served electronically, the documents to be filed under seal shall be filed conventionally, in paper form; and

b. Real Objects:

Exhibits and/or attachments to pleadings or papers that are real objects or which otherwise may not be comprehensively viewed in an electronic format may be filed and served conventionally, in paper form.

Any exhibits designated for conventional filing under subsections 9(i) and (ii) above shall be provided to the Office of the Wayne County Clerk within five (5) days of the corresponding Electronic Filing along with a cover page which describes the related electronically filed documents.

10. Service of Conventionally Filed Pleadings, Papers or Documents

Any conventionally filed pleading, paper or document shall be served on all other parties to the case in accordance with the Michigan Court Rules.

11. Representations by Using a Typographical Signature

Every electronically filed pleading, paper and document shall bear a Typographical Signature of at least one of the attorneys of record, along with the typed name, address, telephone number and e-mail address of that attorney. Typographical Signatures shall be treated as personal handwritten signatures for all purposes contemplated by the Michigan Court Rules.

12. Maintaining Original Affidavits and Returns of Service

The only Electronic Documents or Electronic Images a party is required to maintain in the original, hard copy form are affidavit(s) and/or return(s) of service. Any User who electronically files or serves an affidavit(s) and/or return(s) of service shall make the original of

the Electronic Document and/or Electronic Image available for inspection by the Court or other counsel upon reasonable notice.

13. Time for Filing and Effect of Use of Electronic Filing

Any paper or document filed electronically shall be considered as served and filed with the Office of the Wayne County Clerk when it is completely submitted and receives an authorized date and time. Any paper or document filed electronically before 4:30 p.m. shall be deemed filed for purposes of filing deadlines. Regardless of the location of counsel, Vendor and this Court will apply Eastern Standard Time or Eastern Daylight Time (hereinafter "EST/EDT"), whichever time is in effect in Michigan at the time of the filing of the pleadings, papers and other documents. Thus, for example, if the deadline for filing a paper is March 15, if that paper is filed electronically by 4:30 p.m. EST/EDT on March 15, it will be deemed timely filed. Any document filed after 4:30 p.m. EST/EDT on a day the Office of the Wayne County Clerk is open for business shall be deemed filed on the next day the Office of the Wayne County Clerk is open. Any document filed on a day the Office of the Wayne County Clerk is not open is deemed filed on the next day the Office of the Wayne County Clerk is open. The Vendor is appointed the limited agent of the Wayne County Clerk as to the electronic filing, receipt, service and/or retrieval of any pleading, paper or document with the Vendor. All documents filed conventionally shall comply with the Michigan Court Rules for filing.

Upon receipt and filing of an Electronic Document or Electronic Image, the Vendor shall issue a Confirmation that the document has been received by the Wayne County Clerk. The Confirmation shall serve as proof that the document has been received by the Wayne County Clerk.

A User shall be notified by e-mail of any documents subsequently rejected by the Wayne County Clerk or the Vendor after receipt, and may be required to re-file the document(s) to meet the filing requirement.

14. Problems in Transmission of an Electronic Filing

Users are encouraged by the Court to be reasonable with each other should a technical problem arise which will cause or caused a deadline to be missed by a User. In such an event, Users may, by consent and without the necessity of an order of Court, agree to an extension of a filing deadline. If an agreement cannot be reached, however, and an Electronic Document or Image cannot be filed or was not filed because of the following reasons which are not exclusive: (1) inaccessibility to Vendor; (2) downtime of a User's Internet Service Provider; (3) an error in the transmission of the pleading, paper or document to Vendor which was unknown to the User; or (4) a failure to process the electronic filing when received by Vendor, then the Court may enter such order as is necessary to permit the late filing of the pleading, paper or document.

15. Public Access to the Electronically Filed Pleadings, Papers and Documents

The Wayne County Clerk's office shall make available to members of the general public, without charge and during normal business hours, at least one computer terminal capable of searching and reviewing pleadings, papers and documents filed of public record in the Asbestos Litigation. The Wayne County Clerk shall make copies of any publicly filed Electronic Documents or Electronic Images at a reasonable rate established by the Wayne County Clerk.

D. Form Of Pleadings, Papers And Documents Electronically Filed

1. Format of Electronically Filed Pleadings, Papers and Documents

All Electronic Documents and Electronic Images shall, to the extent practicable, be formatted in accordance with the applicable Michigan Court Rules governing the formatting of

paper pleadings and papers, and in such other and further format as the Court may require from time to time. Pleadings, papers or other documents when electronically filed are digitally converted to a portable document format ("**PDF**") by the Vendor. In the conversion process, a document may be lengthened which may cause such document to exceed a page limit rule which may exist in a given case. As such, the document will be accepted as conforming to the page limit rules as long as the User's own electronic copy of the pleading, paper or document, when viewed, conforms to the page limit restriction.

2. Conversion To Electronic Documents and Electronic Images

Users may either convert documents to Electronic Documents and/or Electronic Images themselves or have the Vendor convert the documents for them. A User choosing to have the Vendor perform the conversion function may forward the documents to the Vendor by mail, express mail or facsimile. The documents forwarded to the Vendor must be in legible form. The Vendor shall convert the documents to electronic form, file the documents with the Court and/or serve the designated parties as directed by the User. Users who file and/or serve their documents through the Vendor shall be charged surcharge fees reflecting the Vendor's then-current published rates for converting, filing and serving in this manner in addition to the Vendor's per page/per document rates. See Exhibit F for the Vendor's contact information.

3. Title of Pleadings, Papers and Other Documents

The title of each Electronic Document or Electronic Image shall include a brief but informative description of the paper. The caption of the Electronic Document or Electronic Image shall also contain the following information:

a. the party or parties filing the paper;

b. the nature of the paper;

c. the party or parties against whom relief, if any, is sought; and

d. the nature of the relief sought, (e.g., "John Doe's Motion to Compel Discovery and for Sanctions against Jim Smith).

The signature page of each Electronic Document or Electronic Image filed shall contain the name of the attorney and, if applicable, the name, address, phone number and e-mail address of the attorney representing the party.

4. Preprinted Caption Label Not Required for Electronic Filing

All Electronic Documents and Electronic Images are excepted from the requirement of LCR 2.114 (C) of bearing a preprinted caption label from the Office of the Wayne County Clerk.

E. Technical Requirements Of Users

1. System Requirements

Vendor shall maintain a list of system requirements and specifications on its website. For the Vendor's website, see Exhibit F.

2. Format

All electronically filed pleadings, papers and documents may be filed in Adobe Acrobat Portable Document Format ("**PDF**") directly, WordPerfect and Microsoft Word formats or other word processing systems that can be converted by the current versions of WordPerfect or Word as of the date of this Order. To ensure all Users will be able to print the retrieved documents from the Vendor correctly, the Vendor converts the Electronic Document to the Adobe Acrobat Reader's PDF.

F. Availability Of Electronically Filed Pleadings, Papers And Documents

Electronic Documents and Electronic Images filed with the Office of the Wayne County Clerk will be available immediately for retrieval on the Vendor's Electronic Filing System.

G. Fees

1. Schedule Of Fees

The fees charged by the Vendor are set forth in the Schedule of Fees attached as Exhibit F, as may be amended from time to time. No fees will be increased by the Vendor without giving at least sixty-three (63) days' prior notice to all Users and approval by the Court.

2. Filing Fees

Any Electronic Document or Electronic Image requiring payment of a filing fee to the office of the Wayne County Clerk in order to achieve valid filing status shall be filed electronically in the same manner as any other electronically filed document.

3. Collection of Fees by Vendor

The Vendor, as limited agent for the Wayne County Clerk, will collect filing fees from Users through direct billing of the User.

4. Transmittal of Fees to Court by Vendor

The Vendor will electronically transmit all filing fees to a financial account designated by the Wayne County Clerk and will electronically provide the Office of the Wayne County Clerk with whatever information it requires for each deposit.

EXHIBIT A

STATE OF MICHIGAN

IN THE CIRCUIT COURT FOR THE COUNTY OF WAYNE

*

Case No. NP
Hon. Robert J. Colombo, Jr.

Plaintiff,

v

*

Defendants.

---

* Plaintiff's Attorney

NOTICE OF COMPLAINT

COMPLAINT AND JURY DEMAND

NOW COME * and * by and through their attorneys, *, and for their Complaint against each Defendant, state as follows:

1. In compliance with Wayne County Circuit Court Judge Robert J. Colombo, Jr.'s Case Management Order of _____, 2003 ("**Order**"), paragraph _____, a Complaint was filed with this Court's "Master File" entitled "Asbestos Master Complaint—Living Plaintiff and Spouse," Standard Pleading No. ___. Plaintiffs adopt by reference each and every allegation in this Master Complaint.

2. Pursuant to the Court's Order, Plaintiffs* and * state as follows:

Living Plaintiff: *

Plaintiff's Residence: *

Spouse: *

Disease: *

Date of Diagnosis: *

Period of Exposure to Asbestos: *

Occupation: *

Employer: *

Social Security Printout included:
(check one) Yes_____ No_____ (has been ordered)

Geographical Situs of Asbestos Exposure:
Wayne County, Michigan

Currently Known Non-Parties Whose Asbestos-Containing Products Plaintiff Has Reason To Believe He Or She Was Exposed To During His Or Her Work Career: *
*
*

A TRIAL BY JURY IS HEREBY DEMANDED.

_____
Plaintiff's Attorney

Date:

IF YOU OR YOUR ATTORNEY DO NOT HAVE A COPY OF THE MASTER COMPLAINT, PLAINTIFF'S ATTORNEY SHALL PROVIDE A COPY UPON REQUEST.

EXHIBIT B

STATE OF MICHIGAN

IN THE CIRCUIT COURT FOR THE COUNTY OF WAYNE

* Personal Representative of the Estate of *, deceased	Case No. NP Hon. Robert J. Colombo, Jr.

Plaintiff,

v

*

Defendants.

---

* Plaintiff's Attorney

NOTICE OF COMPLAINT IN WRONGFUL DEATH CASE

COMPLAINT AND JURY DEMAND

NOW COMES *, Personal Representative of the Estate of *, deceased, by and through her attorneys, * and for her Complaint against each Defendant, states as follows:

1. In compliance with Wayne County Circuit Court Judge Robert J. Colombo, Jr.'s Case Management Order of _____, 2003 ("**Order**"), paragraph _____, a Complaint was filed with this Court's "Master File" entitled "Asbestos Master Complaint—Living Plaintiff and Spouse," Standard Pleading No. ____. Plaintiffs adopt by reference each and every allegation in this Master Complaint.

2. Pursuant to the Order, Plaintiffs* and * state as follows:

Deceased Plaintiff: *

Plaintiff's Residence: *

Spouse: *

Disease: *

Date of Diagnosis: *

Period of Exposure to Asbestos: *

Occupation: *

Employer: *

Social Security Printout included:
(check one) Yes_____ No_____ (has been ordered)

Geographical Situs of Asbestos Exposure:
Wayne County, Michigan

Currently Known Non-Parties Whose Asbestos-Containing Products Plaintiff Has Reason To Believe He Or She Was Exposed To During His Or Her Work Career: *
*
*

A TRIAL BY JURY IS HEREBY DEMANDED.

_____
Plaintiff's Attorney

Date:

IF YOU OR YOUR ATTORNEY DO NOT HAVE A COPY OF THE MASTER COMPLAINT, PLAINTIFF'S ATTORNEY SHALL PROVIDE A COPY UPON REQUEST.

EXHIBIT C

Name:

Initial Data Form

1. **Plaintiff Identification Data:**

   a. **Name:**

   b. **Current Address:**

   c. **Z Number:**

2. **Primary Job Classification as a Seaman:**

3. **Geographical Situs of Asbestos Exposure:**

4. **Wage/Disability/Retirement Information:**

5. **Social Security Printout Included:**
   (check on) Yes _____ No _____ (has been ordered)

6. **Injury, Illness or Disease:**

7. **Date of First Discovery of Illness or Disease:**

8. **Union/Benefit Information:**

9. **Toxin:**

10. **Language:**

11. **Employment History:**

    Started Sailing:

    Employer Name Dates Aboard Vessel Name VSL # Rating

EXHIBIT D

Wayne County Circuit Court
Asbestos – Personal Injury
Trial Date and Discovery Schedule

DEADLINE			
FINAL BROCHURE			
PLAINTIFF EXPERT MEDICAL REPORTS AND MATERIALS ON DEATH CASES			
PLAINTIFF EXPERT MEDICAL REPORTS AND MATERIALS ON LIVING CASES			
PLAINTIFF IME'S BY DEFENDANTS			
DEFENDANT MEDICAL REPORTS AND RETURN OF MATERIALS TO PLAINTIFF			
PLAINTIFFS TO PROPOSE DEPOSITION DATES FOR PLAINTIFFS/PERSONAL REPRESENTATIVES			
DEPOSITIONS OF PLAINTIFFS			
NON-MEDICAL FACT WITNESS DEPOSITION CUT-OFF			
WITNESS LIST EXCHANGE			
NOTICE OF INTENT TO CALL TREATING DOCTORS			
TRIAL DATE			

APPLICABLE TIME OUT PERIODS:

PLAINTIFF TRIAL GROUPS:

EXHIBIT E

CHANGE OF VENUE

Case: No.

Plaintiff's Residence:

Job Sites:

Wayne County_____

Michigan_____

Other_____

Plaintiff's Doctors Reside:

Plaintiff's Hospital:

Non-Expert Witnesses for Plaintiff: Number_____

Wayne County_____

Michigan_____

Other_____

Expert Witnesses for Plaintiff:

Wayne County_____

Michigan_____

Other_____

Medical Records: Number_____

Wayne County_____

Michigan_____

Other_____

Other:

American Thoracic Society Documents

Diagnosis and Initial Management of Nonmalignant Diseases Related to Asbestos

THIS OFFICIAL STATEMENT OF THE AMERICAN THORACIC SOCIETY WAS ADOPTED BY THE ATS BOARD OF DIRECTORS ON DECEMBER 12, 2003

CONTENTS

Asbestos is a general term for a heterogeneous group of hydrated magnesium silicate minerals that have in common a tendency to separate into fibers (1). These fibers, inhaled and displaced by various means to lung tissue, can cause a spectrum of diseases including cancer and disorders related to inflammation and fibrosis. Asbestos has been the largest single cause of occupational cancer in the United States and a significant cause of disease and disability from nonmalignant disease. To this demonstrable burden of asbestos-related disease is added the burden of public concern and fear regarding risk after minimal exposure.

This statement presents guidance for the diagnosis of nonmalignant asbestos-related disease. Nonmalignant asbestos-related disease refers to the following conditions: asbestosis, pleural thickening or asbestos-related pleural fibrosis (plaques or diffuse fibrosis), "benign" (nonmalignant) pleural effusion, and airflow obstruction. This document is intended to assist the clinician in making a diagnosis that will be the basis for individual management of the patient. It therefore provides overarching criteria for the diagnosis, specific guidelines for satisfying these criteria, and descriptions of the clinical implications of the diagnosis, including the basic management plan that should be triggered by the diagnosis. It is understood that disease may be present at a subclinical level and may not be sufficiently advanced to be apparent on histology, imaging, or functional studies.

One of the most important implications of the diagnosis of nonmalignant asbestos-related disease is that there is a close correlation between the presence of nonmalignant disease and the risk of malignancy, which may arise from exposure levels required to produce nonmalignant disease or mechanisms shared with premalignant processes that lead to cancer. The major malignancies associated with asbestos are cancer of the lung (with a complex relationship to cigarette smoking) and mesothelioma (pleural or peritoneal), with excess risk also reported for other sites. There is a strong statistical association between asbestos-related disease and malignancy, but the majority of patients with nonmalignant asbestos-related disease do not develop cancer. On the other hand, the risk of cancer may be elevated in a person exposed to asbestos without obvious signs of nonmalignant asbestos-related disease. However, a diagnosis of nonmalignant asbestos-related disease does imply a lifelong elevated risk for asbestos-related cancer.

DIAGNOSTIC CRITERIA AND GUIDELINES FOR DOCUMENTING THEM

People with past exposure to asbestos consult physicians for many relevant reasons: to be screened for asbestos-related disease, for evaluation of specific symptoms that may relate to past asbestos exposure (known or unsuspected), for treatment and advice, and for evaluation of impairment. In 1986, the American Thoracic Society convened a group of experts to review the literature and to present an authoritative consensus view of the current state of knowledge with respect to diagnosis of nonmalignant disease related to asbestos (2). In 2001, a new group was convened to review and to update the 1986 criteria. This statement constitutes that committee's report, completed in 2004.

The criteria formulated in this statement are intended for the diagnosis of nonmalignant asbestos-related disease in an individual in a clinical setting for the purpose of managing that person's current condition and future health. These general criteria are slightly modified from those presented in 1986 (Table 1) (2):

- Evidence of structural pathology consistent with asbestos-related disease as documented by imaging or histology
- Evidence of causation by asbestos as documented by the occupational and environmental history, markers of exposure (usually pleural plaques), recovery of asbestos bodies, or other means
- Exclusion of alternative plausible causes for the findings

The rest of this statement is largely devoted to presenting clinical guidelines required to document that each of these criteria is met. Demonstration of functional impairment is not required for the diagnosis of a nonmalignant asbestos-related disease, but where present should be documented as part of the complete evaluation. Evaluation of impairment has been exten-

Members of the Ad Hoc Statement Committee have disclosed any direct commercial associations (financial relationships or legal obligations) related to the preparation of this statement. This information is kept on file at the ATS headquarters.

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Internet address: www.atsjournals.org

TABLE 1. CRITERIA FOR DIAGNOSIS OF NONMALIGNANT LUNG DISEASE RELATED TO ASBESTOS

1986 Guidelines	2004 Guidelines	Comparison and Notes
	Evidence of structural change, as demonstrated by one or more of the following:	Demonstrates the existence of a structural lesion consistent with the effects of asbestos. The criteria outlined in the 1986 guidelines were most explicit for asbestosis
Chest film (irregular opacities)	• Imaging methods	Chest film, HRCT, and possibly future methods based on imaging. The 1986 guidelines specified ILO classification 1/1
Pathology (College of American Pathologists)	• Histology (College of American Pathologists)	Criteria for identifying asbestosis on microscopic examination of tissue are unchanged
Consistent time interval	Evidence of plausible causation, as demonstrated by one or more of the following:	Evidence of plausible causation implies that the temporal relationship, including latency, is plausible
Occupational and environmental history	• Occupational and environmental history of exposure (with plausible latency)	
	• Markers of exposure (e.g., pleural plaques)	
Asbestos bodies or fibers in lung tissue	• Recovery of asbestos bodies	The 2004 guidelines are not limited to lung tissue, consider the role of BAL to be established, and deemphasize fibers because they are difficult to detect and a systematic analysis for asbestos fibers is not generally available
Rule out other causes of interstitial fibrosis or obstructive disease	Exclusion of alternative diagnoses	The 1986 guidelines primarily addressed asbestosis but mentioned smoking as a cause of obstructive disease. Implicit in the article, however, is that nonmalignant diseases presenting similarly to asbestos-related disease should also be ruled out
"Evidence of abnormal test"	Evidence of functional impairment, as demonstrated by one or more of the following:	Functional assessment is not required for diagnosis but is part of a complete evaluation. It contributes to diagnosis in defining the activity of disease and the resulting impairment
Crackles, bilateral, not cleared by cough	• Signs and symptoms (including crackles)	Signs and symptoms are not specific for diagnosis but are valuable in assessing impairment
Restrictive disease	• Change in ventilatory function (restrictive, obstructive patterns in context or disease history)	The 1986 criteria admitted the possibility of obstructive disease; the 2004 criteria address this specifically
Reduced diffusing capacity	• Impaired gas exchange (e.g., reduced diffusing capacity)	
	• Inflammation (e.g., by bronchoalveolar lavage)	The 1986 guidelines noted possible utility of bronchoalveolar lavage and gallium scanning but considered them to be experimental techniques. The 2004 guidelines exclude gallium scanning, suggest that additional indicators of active inflammation may become useful in future
	• Exercise testing	

Definition of abbreviations: BAL = bronchoalveolar lavage; HRCT = high-resolution computed tomography; ILO = International Labour Organization.
From References 64 and 65.

sively reviewed elsewhere and is not repeated here (3). Functional impairment may be demonstrated by evidence of symptoms or signs, ventilatory dysfunction, impaired gas exchange, and inflammation. Pulmonary function testing should be conducted in conformity with standards already published by the American Thoracic Society (4, 5), including multiple trials to confirm reproducibility and documentation of all trials attempted.

These guidelines are designed for clinical application, not for research, epidemiologic surveillance, screening, litigation, or adjudication. They balance the need to be as accurate as possible with protection of the patient's safety and the yield, cost, and accessibility of the diagnostic procedures available. These guidelines, if they err, err on the side of specificity rather than sensitivity. This is because nonmalignant asbestos-related disorders are difficult to detect in their earliest stages and because there is no early intervention that has been proven to alter the subsequent evolution of the disease. On the other hand, the documentation of causation by asbestos carries important implications for the patient and can be established with reasonable certainty, once the disease is identified.

Asbestos as a Hazard

The generic term "asbestos" is used to describe a group of minerals that, when crushed, break into fibers. As defined by the National Research Council (1), the term "asbestos" is a "commercial-industrial term rather than a mineralogical term. It refers to well-developed and hair-like long-fibered varieties of certain minerals that satisfy particular industrial needs." They are chemically heterogeneous hydrated silicates and each has chemical analogs with different structures that do not form fibers. Fibers have parallel sides with length three or more times greater than width. Asbestos fibers have great tensile strength, heat resistance, and acid resistance; varieties are also flexible. The six minerals that are traditionally defined as asbestos include chrysotile asbestos (the asbestiform variety of serpentine); the amphiboles, which include crocidolite (the asbestiform variety of riebeckite) and amosite (the asbestiform variety of cummingtonite-grunerite); and the asbestiform varieties of the amphiboles, which include anthophyllite (anthophyllite asbestos), actinolite (actinolite asbestos), and tremolite (tremolite asbestos) (6). Just as all forms of asbestos, by the definition and classification above, appear to cause malignancy, all may cause the nonmalignant diseases described. Issues of relative potency among the forms of asbestos, and particularly between chrysotile and the amphiboles, are primarily of concern with respect to the risk of malignancy and are not discussed in this document.

Commercial-grade asbestos is made up of fiber bundles. These bundles, in turn, are composed of extremely long and thin fibers, often with splayed ends, that can easily be separated from

one another. Commercial asbestos has high tensile strength, flexibility, resistance to chemical and thermal degradation, and high electrical resistance, and can often be woven. On the basis of these characteristics, asbestos was broadly used in the past in insulation, brake linings, flooring, cement, paint, textiles, and many other products; however, commercial use has declined substantially in more recent years.

Asbestos and asbestiform minerals may occur as a natural accessory mineral in other industrial mineral deposits or rocks. These asbestiform amphiboles and some other fibrous minerals may not completely fit the commercial definition of asbestos but may have similar effects, such as the tremolite-like asbestiform mineral found in association with vermiculite in Libby, Montana (7). Although the general criteria still apply, the specific diagnostic guidelines provided in this statement may or may not apply in such situations, depending on the mineral and exposure circumstances. Documentation of health effects in the scientific literature for these minerals is not as extensive as for chrysotile and the common amphiboles.

World production and use of asbestos climbed steadily since its commercial introduction in the late nineteenth century and fell rapidly after documentation of its hazards in the 1970s and 1980s. In Western industrialized countries, the widespread use of asbestos in industry and in the built environment in the first seven decades of the twentieth century has resulted in an epidemic of asbestos-related illness that now continues into the twenty-first century, despite decline in global production and use. Its use has now been banned in many Western countries. Asbestos is still mined in Russia and China, mainly for local use, and in Canada, where most of the product is exported to Asia and Africa.

Today, with stringent regulation of asbestos use and the disappearance of almost all asbestos-containing products from the market, nonmalignant asbestos-related disease is primarily a concern in four settings in the developed world: (*1*) the historical legacy of asbestos exposure affecting older workers; (*2*) the current risk experienced by the workforce engaged in certain occupations managing the remaining hazard, such as building and facility maintenance; (*3*) asbestos abatement operations, removing insulation and other asbestos-containing products; and (*4*) renovation and demolition of structures containing asbestos. In the developing world, workers and their families continue to be exposed. In some countries, including industrialized countries formerly belonging to the Eastern bloc and rapidly industrializing countries in Asia, the use of asbestos continues and may even be increasing.

Asbestos is still a hazard for an estimated 1.3 million workers in the construction industry in the United States and for workers involved in maintenance of buildings and equipment (8). Most asbestos in the United States today exists in building and machinery insulation and old products, such as appliances, that may be available for resale. New products that may contain asbestos today in the United States include friction surfaces (brake pads), roofing materials, vinyl tile, and imported cement pipe and sheeting. Significant asbestos content may be present as a contaminant in vermiculite insulation often found in homes (7).

Historically, occupations at greatest risk for nonmalignant asbestos-related disease have tended to be those engaged in the production and end use of products made from asbestos. These have included a wide assortment of items, including friction pads, brake linings, gas masks, cement water pipe, insulation, and textiles. Occupations engaged in the mining and extraction of asbestos have usually shown lower frequencies of nonmalignant asbestos-related disease. Passive exposure, including workers carrying home asbestos on their clothing, was historically associated with elevated cancer risk, particularly mesothelioma, and risk of nonmalignant asbestos-related disease. Workers in building and equipment maintenance may still encounter asbestos insulation even though asbestos is no longer widely used in commerce. Asbestos abatement activities, including removal and replacement of insulation, provide opportunities for exposure among contemporary workers (8).

Asbestos in Lung Tissue

Asbestos fibers carried to the deep lung induce an alveolitis that results in fibrosis. Inhaled asbestos fibers can also result in pleural inflammation. Asbestos fibers are transported to the pleural surface along lymphatic channels by macrophages and/or by direct penetration. The degree of fibrosis in asbestosis is dose dependent (9–12).

Asbestos fibers are deposited at airway bifurcations and in respiratory bronchioles and alveoli primarily by impaction and interception. Fibers migrate into the interstitium, in part via an uptake process involving Type I alveolar epithelial cells. This causes an alveolar macrophage–dominated alveolitis, as demonstrated in Figure 1 (12, 13). Thereafter, many of the fibers are cleared.

Activated macrophages are stimulated to engulf and remove asbestos fibers. This process is not uniformly successful, however, and many fibers are retained (9, 10). The long fibers cannot be completely engulfed by the macrophage, as demonstrated in Figure 2.

Chrysotile fibers also split longitudinally, creating additional fibrils. These are cleared more efficiently than amphibole asbestos fibers, which may be retained indefinitely (12). The fibers induce apoptosis, a form of controlled cell death, in the macrophage and stimulate inflammation. This effect is reduced once the fiber is coated to create an asbestos body, but the great majority of fibers in the lung remain uncoated. For these reasons, asbestos has a prolonged residence in the lung, penetrates the interstitium of the distal lung, and shows extensive mobility both in the lung and around the body (9).

Asbestos fibers, in particular, stimulate macrophages to produce a variety of mediators. Oxygen radicals contribute to tissue injury. Granulocytes are recruited to sites of disease activity and they in turn release mediators that contribute to tissue fibrosis by stimulating fibroblast proliferation and chemotaxis and ultimately promoting collagen synthesis (11–15).

The inflammatory processes induced by asbestos include alveolitis, inflammation in the surrounding interstitium, and inflammation followed by fibrotic change in the respiratory bronchioles that extends into adjacent alveolar tissue (11, 14, 16). Studies of the lung tissue of asbestos-exposed workers, including nonsmokers, have demonstrated a form of peribronchiolitis involving the walls of membranous and respiratory bronchioles, that shows characteristics of a more intense fibrotic response than the small airway lesions caused by nonspecific mineral dusts that the lesions otherwise resemble (17, 18).

Asbestos fibers and their derivatives, asbestos bodies, can be identified and quantified in lung tissue and bronchoalveolar lavage (BAL) specimens, as demonstrated in Figure 2 (19). Transbronchial lung biopsy is less reliable than BAL or open lung biopsy in recovering sufficient tissue to demonstrate elevated asbestos body or fiber counts when they do occur (20).

Asbestos fibers, unlike asbestos bodies, are rarely seen by light microscopy and must be analyzed by scanning/transmission electron microscopy (19, 21, 22). There is considerable variation among laboratories in procedures to quantify asbestos fibers in tissue (18, 23, 24), which has led to efforts to standardize procedures (19). Asbestos mineralogical types can be identified by energy-dispersive X-ray analysis, in which detection of magnesium and silicon is characteristic of most forms of asbestos and

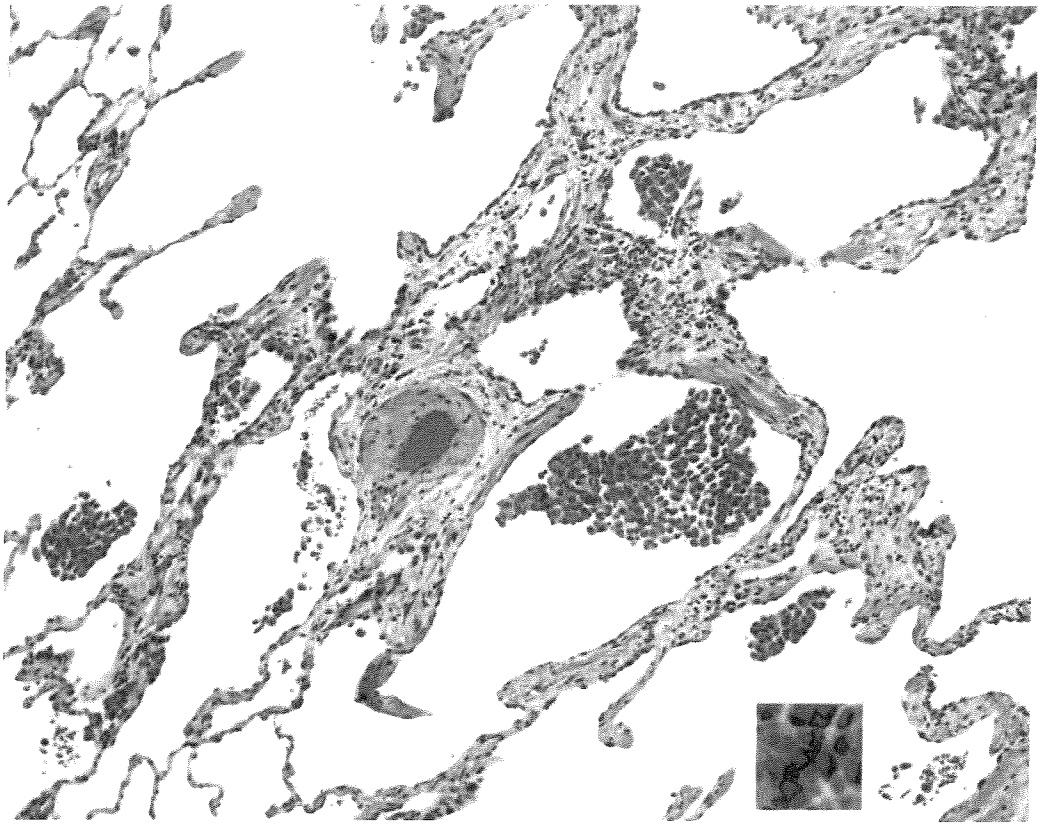


Figure 1. Low-power photomicrograph of hematoxylin and eosin (H&E)-stained sections from a patient with asbestosis, showing patchy asbestosis and a moderate number of macrophages within the alveoli. *Inset*: Close-up of macrophages in an iron-stained section showing an asbestos body.

the presence of a large iron peak signifies an amphibole (with the exception of tremolite) (25). Fiber analysis can be helpful in assessment of exposure and provides information about intensity, duration, and latency (e.g., uncoated fibers may reflect recent heavy exposure). However, because some fibers dissolve over time, the absence of a high fiber count does not necessarily mean that there has been no exposure, especially when chrysotile is the predominant exposure (22). Mineralogic analysis of asbestos fibers is largely a research technique and is not widely available (26).

Asbestos bodies. Asbestos bodies are asbestos fibers that have

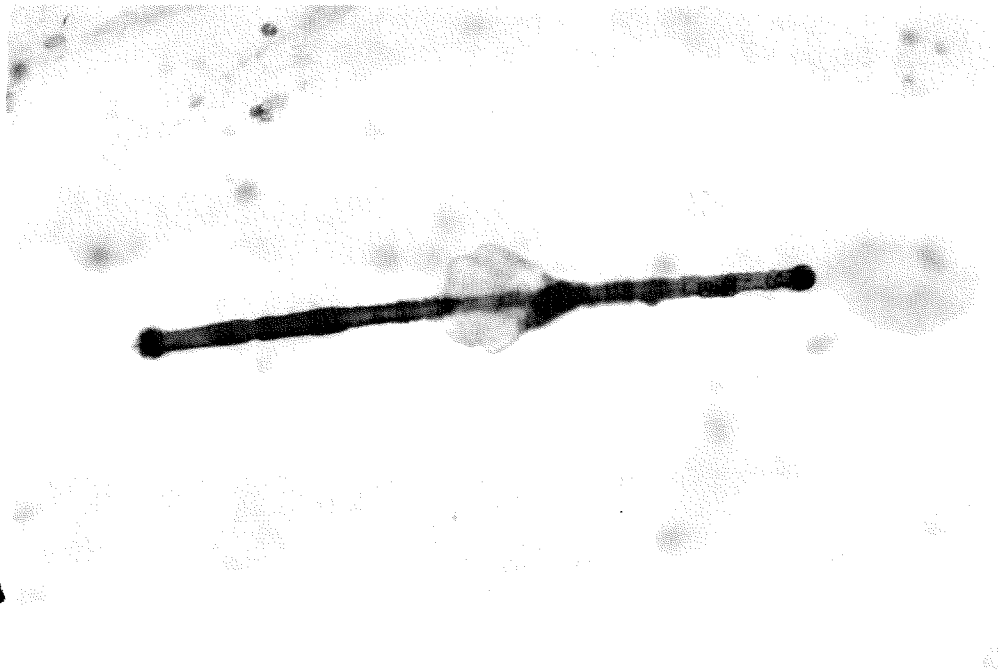


Figure 2. Asbestos body retrieved by bronchoalveolar lavage. Note its clear central core.

been coated with an iron-rich, proteinaceous concretion (Figures 1 and 2). Amphibole asbestos forms the majority of asbestos bodies and is more persistent in lung tissue than chrysotile (25). Asbestos bodies are larger than asbestos fibers and can be identified and quantified by light microscopy. An iron stain is helpful to identify fibrous bodies coated by iron (hence the general name "ferruginous bodies"). Ferruginous bodies generally form on fibers at least 10 μm in length, and more than 90% of all coated fibers have asbestos cores. Demonstration of an elevated body burden of asbestos confirms past exposure (19). Levels of at least one or two asbestos bodies per field of a tissue section on a slide under light microscopy are consistent with occupational exposure (19, 22, 24).

Transbronchial biopsy. Transbronchial lung biopsies are usually too small to analyze for asbestos bodies. Bronchoalveolar lavage recovers more material and therefore provides a better indicator of tissue burden. Some experienced clinicians have found that identification of six or more bodies in bleach-digested samples from at least two biopsies is characteristic of patients with occupational exposure (26). However, the absence of observable asbestos bodies is not reliable in excluding significant exposure in transbronchial biopsy tissue (20).

These indicators of fiber burden are sufficient but not necessary to identify occupational exposure and to diagnose asbestos-related disease. Beyond clinical research, the method has applications in litigation and exposure assessment for epidemiology.

Bronchoalveolar lavage. Asbestos bodies and fibers can be identified and quantified in BAL specimens, as in Figure 2 (22). There is considerable variation among laboratories in these tests (18, 19, 22, 23). The count of asbestos bodies in BAL fluid appears to correlate with the presence or degree of fibrosis in some studies but not others (24, 27, 28).

BAL in patients with asbestosis has demonstrated an alveolar macrophage alveolitis associated with a modest increase in neutrophils (12, 13). This neutrophilia correlates with the finding of crackles (rales) on physical examination and disturbances in oxygenation (12, 27) and is apt to be more pronounced in patients with advanced disease (13). Clinically apparent asbestosis occurs only after a significant latent period. However, studies using BAL, computed tomography (CT) scanning, and gallium-67 scanning have demonstrated that inflammatory events occur well before the onset of clinical disease. Thus, it is likely that the initial exposure induces inflammation and injury that persist through the latent or subclinical phase and later develop into the clinical disease, which is typically diagnosed by chest imaging (13).

CLINICAL EVALUATION AND INDICATORS

The clinical evaluation of nonmalignant asbestos-related disease should consider subjective symptoms as well as objective findings on physical examination, pulmonary function tests, and chest radiographic studies. In the large majority of patients, the diagnosis of nonmalignant asbestos-related lung disease is based on the clinical findings discussed below, in the context of an appropriate history of exposure to asbestos and a documented latency period sufficient to place an individual at risk.

Symptoms

The insidious onset of dyspnea is the most common respiratory symptom associated with asbestosis, typically beginning with dyspnea on exertion. A nonproductive cough is commonly present. The presence of wheeze or dyspnea (27), as reported on the ATS-DLD-78A respiratory questionnaire (5), is strongly associated with diminished ventilatory capacity in cross-sectional studies of asbestos-exposed workers, with an 11 to 17% reduction in ventilatory capacity (27, 29). A 2–8% reduction in ventilatory capacity has been observed for cough, phlegm, and symptoms of chronic bronchitis among asbestos-exposed workers (29). Development or progression of respiratory symptoms has been associated with accelerated loss of ventilatory capacity in a longitudinal investigation of asbestos-exposed workers, with an excess 28-ml/year decline in FEV_1 associated with development of dyspnea, and 67-ml/year excess decline in FVC associated with newly developed wheezing, relative to asymptomatic individuals (30).

In a study of 64 patients, diffuse pleural thickening or fibrothorax was associated with dyspnea on exertion, usually mild, in 95%, chest pain in more than half, and restrictive defect in one-third. The chest pain was intermittent in most but constant in 9% (31). Rapidly progressive or severe chest pain should raise clinical suspicion of either malignancy or a nonmalignant pleuritis.

Subjective symptoms are not easily interpreted in the absence of objective findings but provide important ancillary information. The persistence or new onset of respiratory symptoms is correlated with accelerated loss of lung function in asbestos-exposed workers and therefore may predict future risk (30).

Occupational and Environmental History

It is essential to take a comprehensive occupational and environmental history when asbestos-related disease is suspected (32). The occupational history should emphasize occupational and environmental opportunities for exposure that occurred about 15 years and more before presentation.

The diagnosis of asbestosis is ideally based on an accurate exposure history, obtained whenever possible directly from the patient, that defines the duration, intensity, time of onset, and setting of exposure experienced by the patient. Patients may forget short periods of employment, during which intense exposure is possible, or employment early in their lives. In such cases the characteristic radiographic signs of asbestos exposure may be enough to document exposure.

The occupational title is not enough, as the names of many occupations and trades are uninformative, such as "millwright" or "fireman" (a misleading title that sometimes refers to furnace workers and stokers) or "mixer." Representative occupational exposures include, but are not limited to, manufacture of asbestos products, asbestos mining and milling, construction trades (including insulators, sheet metal workers, electricians, plumbers, pipefitters, and carpenters), power plant workers, boilermakers, and shipyard workers.

Asbestosis is commonly associated with prolonged exposure, usually over 10 to 20 years. However, short, intense exposures to asbestos, lasting from several months to 1 year or more, can be sufficient to cause asbestosis. For example, shipyard workers who applied or removed insulation in confined spaces have developed asbestosis after brief periods of heavy exposure. Insulation workers have had similarly intense exposures during their apprenticeship when they unloaded asbestos-containing sacks into troughs for mixing asbestos cement. Such occupational exposures are now rare but were common in the United States from the years after World War II until the 1970s. Adequate industrial hygiene controls were absent or not widely applied. Protective regulations were inadequate and only partially enforced during much of that period.

Workers whose own jobs may not require handling asbestos may still be "bystanders" who worked in close proximity to other users, especially in the construction trades, where workers have experienced exposure from insulation being installed around them. Among sheet metal workers, for example, the prevalence of asbestos-related changes on chest film was 31% (19% pleural only, 7% parenchymal only, and 6% both). Among those who had been in the trade for 40 or more years, 41.5% had radio-

graphic findings (33). These findings established that sheet metal workers, although not working directly with asbestos, had substantial exposure in the work environment.

Measures taken to protect workers, or lapses in these measures, may be important in documenting exposure. Although exposure levels are generally low in developed countries today, lapses occur and were more frequent in the past. Some patients who have immigrated may have worked in countries where occupational health regulations have been poorly enforced or where environmental exposure has occurred.

Environmental sources of exposure, for example, tailings of asbestos mines or prolonged exposure in buildings with exposed sources of asbestos contamination, may be important in some cases. Passive exposure, for example, of children in the home when asbestos is brought into the house on the clothes of a worker, may cause disease (34). Undisturbed and nonfriable asbestos insulation in buildings, including schools, does not present a hazard.

The prevalence of asbestosis among asbestos workers increases with the length of employment, as illustrated in an early report in which investigators analyzed chest films of 1,117 New York and New Jersey asbestos insulation workers. They found asbestosis in 10% of workers who had been employed for 10 to 19 years, 73% among those employed for 20 to 29 years, and in 92% of those employed for 40 or more years (35). A similar exposure–response relationship was found among asbestos cement workers (36).

Differences in solubility among the various types of asbestos may affect fiber retention, body burden, and the risk of nonmalignant disease. The clinician is rarely in a position to evaluate this aspect of exposure and there is no validated means to adjust the occupational history to take this factor into account. Solubility is primarily of concern with respect to projecting future risk, particularly of malignant disease, given a history of exposure. It is irrelevant to diagnosis when disease is already present and other indicators of exposure are demonstrable.

Physical Examination

Physical findings in asbestosis include basilar rales, often characterized by end-inspiratory crackles (rales) (36, 37); in some cases of advanced asbestosis, finger clubbing may be present. Physical findings of crackles, clubbing, or cyanosis are associated with increased risk for asbestos-related mortality (36). Although these physical signs are useful when present, their overall clinical utility is limited by low sensitivity. For example, in one study as many as 80% of individuals with radiographic asbestosis demonstrated crackles, a frequency that appears to be unusually high in the experience of other clinicians (27).

Conventional Imaging

The chest radiograph remains an extremely useful tool for the radiographic diagnosis of asbestosis and asbestos-related pleural disease, and is widely available internationally. The plain film has long been the basis for assessing asbestos-related disease of the lung and pleura. A standardized system for taking and classifying films for presence and profusion of opacities consistent with pneumoconiosis and for pleural changes was developed in the 1950s and is now known as the *International Classification of Radiographs of Pneumoconiosis* (or "ILO classification" after its sponsor, the International Labour Organization). The ILO classification has been revised (38). This system, which is the basis of the "B-reader" qualification for designating persons as competent in classifying pneumoconiosis films, was developed for grading the radiographic severity of pneumoconiosis in epidemiologic studies but has been applied to clinical settings to maintain consistency in classifying chest films. The ILO classification requires conventional film-based posteroanterior (PA) chest films taken at prescribed specifications and classified with due regard for quality. Conventions for classifying digitized films and other advanced imaging systems have lagged behind the development of technology.

The initial radiographic presentation of asbestosis is typically that of bilateral small primarily irregular parenchymal opacities in the lower lobes bilaterally. Over time, the distribution and density or "profusion" of opacities may spread through the middle and upper lung zones. Although irregular opacities are most common from asbestos exposure, mixed irregular and rounded opacities are often present. The ILO classification profusion score correlates strongly with mortality risk (36), reduced diffusing capacity, and diminished ventilatory capacity (37, 39). A critical distinction is made between films that are suggestive but not presumptively diagnostic (0/1) and those that are presumptively diagnostic but not unequivocal (1/0). This dividing point is generally taken to separate films that are considered to be "positive" for asbestosis from those that are considered to be "negative." However, profusion itself is continuous (36, 38).

Plain chest radiographs are limited with respect to sensitivity and specificity in cases of mild or early asbestosis. Among individuals with asbestosis confirmed by histopathologic findings, 15–20% had no radiographic evidence of parenchymal fibrosis in one study (40), similar to the proportion of other interstitial lung diseases that present with normal chest films (41).

Pleural plaques are frequently documented on plain chest radiographs, but CT is more sensitive for their detection. Only 50 to 80% of cases of documented pleural thickening demonstrated by autopsy, conventional CT, or high-resolution CT (HRCT) are detected by chest radiograph (42, 43). Plain chest radiographs are also limited by specificity in cases of mild pleural disease, which may be difficult to distinguish from extrapleural fat pads (39, 44). Oblique views can enhance both sensitivity and specificity of plain chest radiographs in clinical settings where HRCT is unavailable, but may also fail to distinguish plaques from fat pads (45). CT and HRCT are discussed in the next section.

Computed Tomography

A chest film clearly showing the characteristic signs of asbestosis in the presence of a compatible history of exposure is adequate for the diagnosis of the disease: further imaging procedures are not required. Conventional CT is superior to chest films in identifying parenchymal lesions, rounded atelectasis, and pleural plaques (46). However, conventional CT has been displaced by HRCT for the evaluation of asbestos-exposed subjects because the latter is more sensitive for detecting parenchymal fibrosis.

In subjects with low profusion categories of asbestosis, CT signs tend to be clustered as follows (47):

- Honeycombing and thickening of septa and interlobular fissures, suggesting interstitial fibrosis
- Diffuse pleural thickening, parenchymal bands, and rounded atelectasis, suggesting diffuse fibrosis involving the visceral pleura
- Pleural plaques

HRCT has an important role when experienced readers disagree about the presence or absence of abnormalities on a high-quality chest film, when chest radiographic findings are equivocal, when diminished pulmonary function is identified in association with otherwise normal plain chest radiographic findings, and when extensive overlying pleural abnormalities do not allow a clear interpretation of parenchymal markings. Because HRCT is more sensitive than other techniques for detecting parenchymal changes, it may reveal abnormalities with uncertain prognostic

significance. HRCT is more specific than plain chest radiographs, excluding conditions such as emphysema, vessel prominence, overlying pleural disease, and bronchiectasis, which may confound radiographic interpretation.

HRCT is much more sensitive in the detection of asbestosis than plain chest radiographs (46, 48), although even a normal HRCT cannot completely exclude asbestosis (49). Among asbestos-exposed individuals with unremarkable chest radiographic findings (ILO score 0/0 or 0/1), 34% were identified by HRCT as having findings suggestive of asbestosis. HRCT findings also correlated with decrements in pulmonary function tests in these cases, with a significantly diminished vital capacity and diffusing capacity (50).

HRCT can detect early pleural thickening (i.e., 1–2 mm in thickness) much more sensitively than plain chest radiographs. Pleural thickening is frequently discontinuous and interspersed with normal regions. It is usually bilateral but may be unilateral in a third of cases (48). HRCT also offers an advantage over plain chest radiographs in specificity, being able to distinguish pleural disease from extrapleural fat (51).

HRCT should be obtained at 2-cm intervals, to allow a more accurate assessment of pleural abnormalities, as well as other abnormal findings such as pulmonary masses (52). Prone views should always be obtained, as it is essential to distinguish between dependent atelectasis and parenchymal fibrosis in the posterior lung fields. HRCT findings in asbestosis are typically bilateral, and include evidence of fibrosis (e.g., intralobular interstitial thickening and interlobular septal thickening), subpleural "dotlike" opacities, subpleural lines, parenchymal bands, occasionally ground-glass opacity, and honeycombing in advanced disease (47, 52, 53). A proposal has been put forward for a classification system analogous to that of the ILO system for plain chest radiographs (54), but none has been widely adopted.

The extent of plaque formation does not correlate with cumulative asbestos exposure and thus cannot be used to estimate exposure (55).

Bronchoalveolar Lavage

Sputum analyses for asbestos bodies miss almost half of occupationally exposed individuals in whom asbestos bodies are found on BAL (56). Thus, on the rare occasions in which the diagnosis of asbestosis hinges on demonstration of asbestos bodies and fibers to document exposure, BAL should be performed if sputum analysis is negative (19). Subjects with long-term exposure have higher concentrations of fibers than those with more recent exposure, probably because of higher workplace exposures in the past (19).

Asbestos bodies (ABs) in BAL fluid correlate with occupational exposure and asbestosis (10, 19, 56, 57) and with asbestos bodies in the lung (57). Patients with asbestosis consistently have 2 to 5 orders of magnitude more ABs per milliliter than do pleural plaque subjects. Recovery of more than 1 AB/ml indicates a high probability of substantial occupational exposure to asbestos (19, 58). In one large series, patients with asbestosis had a log mean of 120 AB/ml, those with pleural plaques had 5 AB/ml, those exposed to asbestos who had a normal chest X-ray had 4 AB/ml, and those with malignant mesothelioma or lung cancer had 8 AB/ml. Of those with more than 100 AB/ml, 60% had asbestosis; others had pleural plaques, mesothelioma, or lung cancer, and only 6% were exposed but had no evidence of pathology (59).

BAL cells can also be digested with bleach and the residue analyzed by electron microscopy, with fibers expressed per 10^6 alveolar macrophages (58). In U.S. asbestos insulation workers, electron microscopy identified 1 chrysotile fiber in every 35 alveolar macrophages and 1 amosite fiber per 215 macrophages, with no crocidolite detected. BAL performed on asbestos-exposed subjects has recovered 28 $\times 10^3$ fibers compared with 1 $\times 10^3$ in unexposed subjects (60). For every 100 fibers, there is typically 1 asbestos body (61). Clinically, the appearance of fibers or beaded fibers on a single centrifuged BAL sample mounted on a Diff-Quik slide represents an indicator of parenchymal asbestosis (28).

Amphibole fiber recovery on BAL correlates well with amphibole fiber burden in the lung, but the relationship does not hold for chrysotile because of translocation, clearance, and dissolution (57, 61–63).

Pulmonary Function Tests

Evaluation of subjects with suspected asbestos-related disease should include spirometry (with a hard copy of the flow–volume loop for the permanent medical record), all lung volumes, and the carbon monoxide diffusing capacity. Care should be taken to discriminate among effects due to asbestosis, chronic obstructive pulmonary disease, and restrictive changes due to obesity.

As with other interstitial lung diseases, the classic finding in asbestosis is a restrictive impairment. Mixed restrictive and obstructive impairment is frequently seen; isolated obstructive impairment is unusual. Restrictive impairment may also be observed with pleural disease (*see* section on pleural abnormalities below).

In addition to diminished lung volumes, the carbon monoxide diffusing capacity is commonly reduced due to diminished alveolar–capillary gas diffusion, as well as ventilation–perfusion mismatching. Although a low diffusing capacity for carbon monoxide is often reported as the most sensitive indicator of early asbestosis, it is also a relatively nonspecific finding.

Exercise testing is generally not required for diagnostic purposes, but may be useful in assessing aerobic work capacity in selected cases, or when the degree of dyspnea correlates poorly with objective pulmonary function measurements.

NONMALIGNANT DISEASE OUTCOMES

Asbestosis

Asbestosis is the interstitial pneumonitis and fibrosis caused by inhalation of asbestos fibers. After asbestos exposure, asbestosis becomes evident only after an appreciable latent period. The duration and intensity of exposure influence the prevalence of radiographically evident parenchymal pulmonary fibrosis. In work sites around the world that meet recommended control levels, high exposure to asbestos is now uncommon and clinical asbestosis is becoming a less severe disease that manifests itself after a longer latent interval.

Asbestosis specifically refers to interstitial fibrosis caused by the deposition of asbestos fibers in the lung (Figure 3). It does not refer to visceral pleural fibrosis, the subpleural extensions of fibrosis into the interlobular septae or lesions of the membranous bronchioles.

The College of American Pathologists has developed histologic criteria for asbestosis and a grading system to describe the severity and extent. The mildest (Grade I) form of asbestosis involves the alveolated walls of respiratory bronchioles and the alveolar ducts (Figures 4 and 5). More severe histologic grades involve greater proportions of the acinus (Grade II) until the whole acinar structure is involved (Grade III asbestosis) and some alveoli are completely obliterated (Figure 5). Alveolar collapse, with fibrosis and honeycomb remodeling resulting in new dilated spaces in the parenchyma, results in the most severe grade of asbestosis (Grade IV) (64, 65) (Figure 6). These patterns of acinar fibrosis together with the demonstration of asbestos bodies in standard histologic sections are diagnostic of asbestosis.

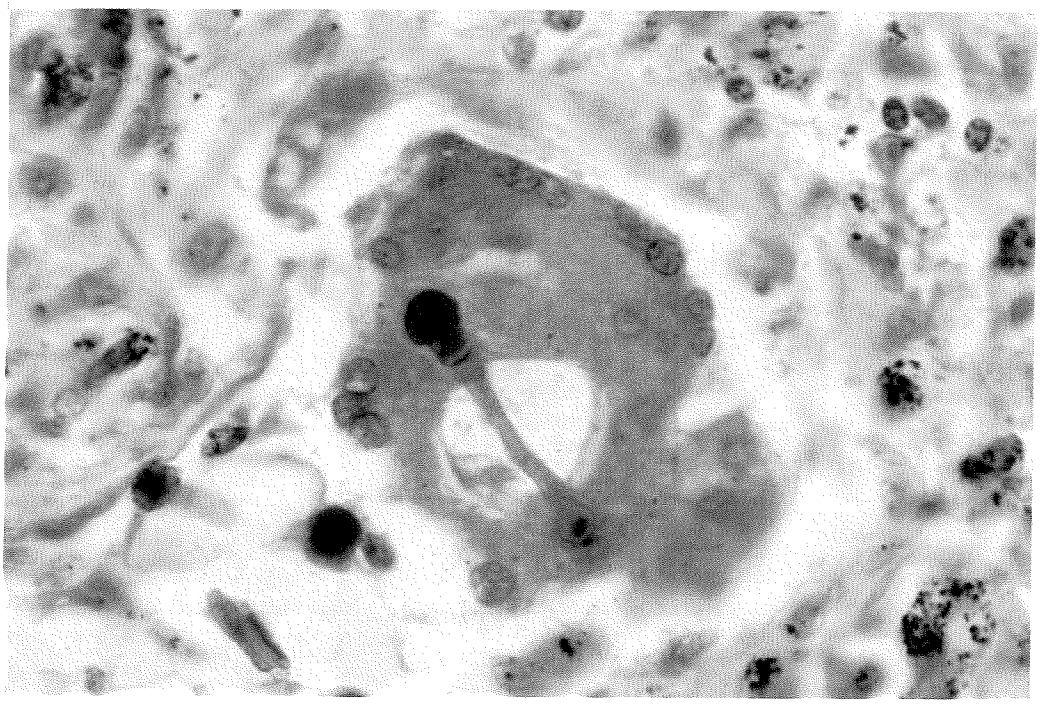


Figure 3. H&E-stained section demonstrating asbestos bodies within alveolus of person with asbestosis. At *center* is a single large asbestos body within a multinucleated giant cell.

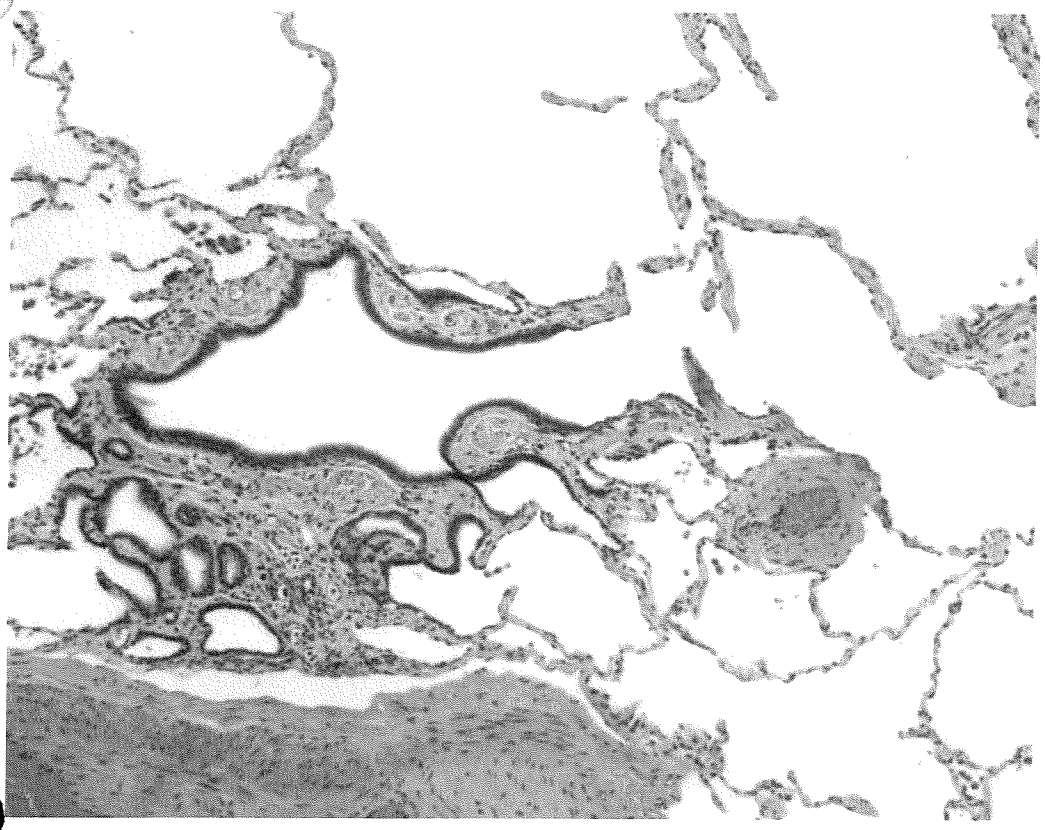


Figure 4. H&E-stained section showing junction of terminal (membranous) bronchiole with a respiratory bronchiole from a person with asbestosis who was an ex-smoker. The walls of the bronchioles are thickened by collagen and show mild smooth muscle hyperplasia. There is a mild chronic inflammatory cell infiltrate in the wall. These features are consistent with asbestos-related small airway disease.

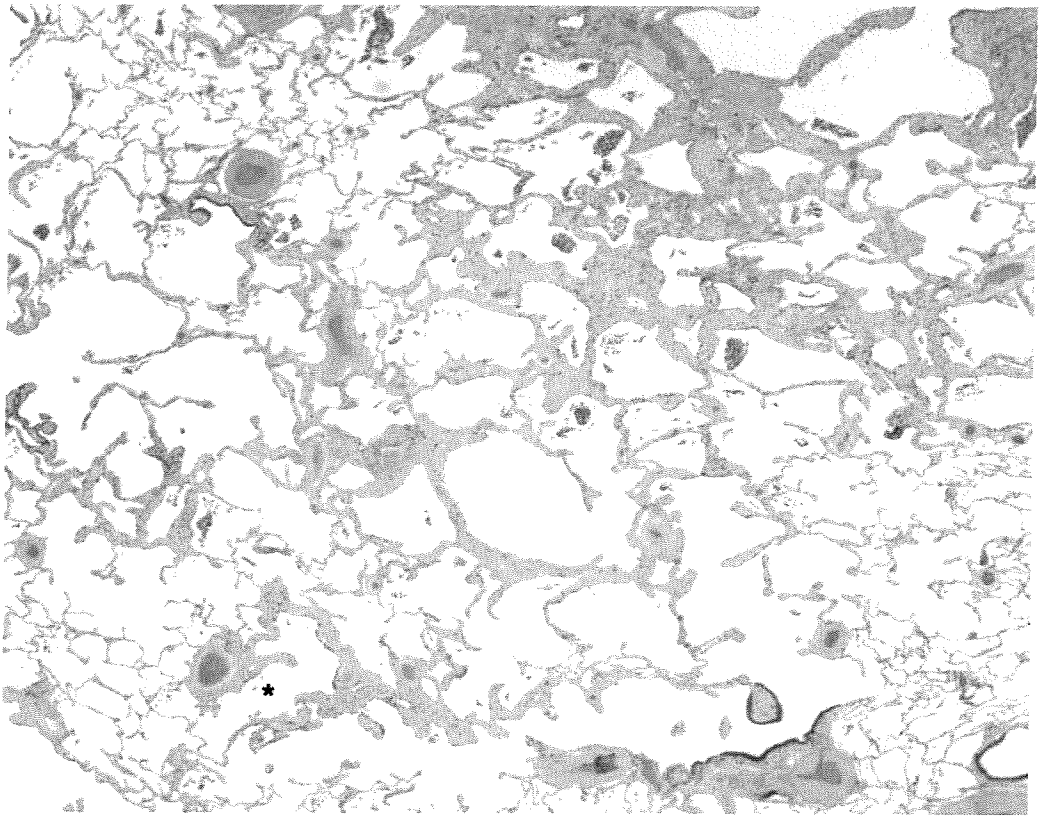


Figure 5. Photomicrograph showing predominantly Grade III asbestosis, partially defined by diffuse interstitial fibrosis extending from acinus to acinus. The respiratory bronchiole at *bottom left* (*) could be classified as a Grade I lesion (*see* Table 2).

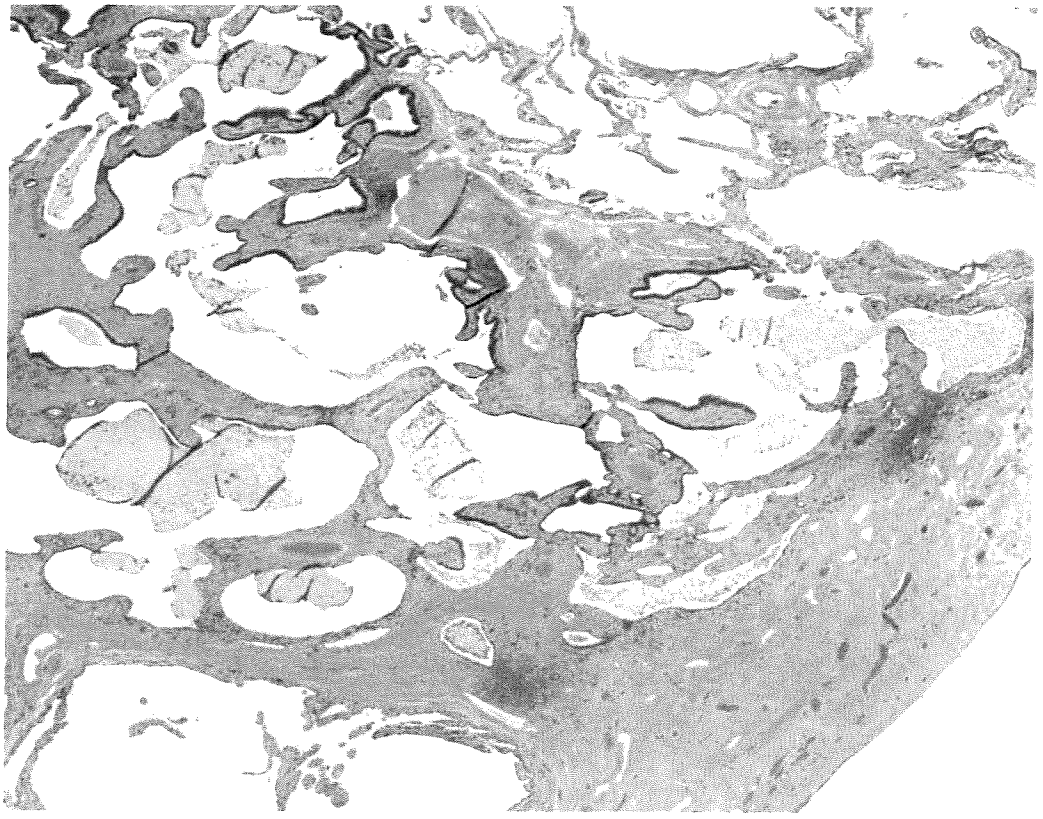


Figure 6. H&E-stained section of lung showing Grade IV asbestosis with honeycombing. The overlying pleura (*bottom right*) is also thickened.

TABLE 2. HISTOLOGIC GRADES OF ASBESTOSIS

Grade	Change
Grade of severity	
0	No fibrosis associated with bronchioles
1 or I	Early fibrosis involving walls of at least one respiratory bronchiole, with or without extension into septa of adjacent alveoli; fibrosis confined to alveolated walls of respiratory bronchioles and ducts and not present in more distant alveoli. Alveolitis and inflammation similar to that caused by cigarette smoking
2 or II	More severe fibrosis involving acinus: alveolar ducts and/or two or more layers of adjacent alveoli. Normal lung remains in a zone between adjacent bronchioles
3 or III	Fibrosis advanced and coalescent, involves entire acinus; all lung between at least two adjacent bronchioles is affected. Some alveoli are completely obliterated
4 or IV	Honeycomb remodeling and large (up to 1 cm) dilated spaces grossly visible in parenchyma
Grade of extent	
A or 1	Only occasional bronchioles are involved. Most appear normal
B or 2	"More than occasional" but less than half of bronchioles are involved
C or 3	More than half of bronchioles are involved

Developed in 1980 by a committee of the College of American Pathologists.

Iron stains may facilitate recognition of the asbestos bodies; however, the presence of asbestos bodies alone is not sufficient to establish the diagnosis of asbestosis. Asbestosis is associated with a variable degree (usually mild) of chronic inflammation and increased numbers of alveolar macrophages, including multinucleate giant cells. The grades of asbestosis correlate with counts and frequencies of asbestos fibers and bodies in the lung and estimates of cumulative workplace exposure (12, 66) (Table 2).

Only the more severe grades of asbestosis are detectable by gross examination. In its classic form, there is diffuse, bilateral, pale, firm fibrosis most severe in the peripheral zones of the lower lobes. Honeycomb cysts and areas of confluent fibrosis may be present (Figure 7). Milder forms of asbestosis and asbestos-associated small airway disease may not be apparent to gross inspection or to palpation, hence the importance of adequate sampling for histology. This should include peripheral and central areas of all lung lobes (depending on the specimen) as well as portions of visibly diseased lung. Adequate sampling of lung adjacent to resected tumors is particularly important and frequently overlooked or inadequately sampled by pathologists. It is strongly recommended that, when biopsy is performed, thoracic surgeons specifically request additional sampling of lung parenchyma in resected lung specimens from patients with known or suspected asbestos exposure (64, 65).

Asbestosis is more prevalent and more advanced for a given duration of exposure in cigarette smokers, presumably because of reduced clearance of asbestos fibers in the lung (67). Some studies suggest that smokers without dust exposure may show occasional irregular radiographic opacities on chest film, but if so the profusion is rarely as high as 1/0; smoking alone therefore does not result in a chest film with the characteristics of asbestosis (68). Both smokers and ex-smokers have a higher frequency of asbestos-related irregular opacities on their chest radiographs than do nonsmoking asbestos-exposed workers in all profusion categories (68–70). Smoking does not affect the presentation of asbestos-related pleural fibrosis.

Clinical diagnosis. Asbestosis is asbestos-induced pulmonary parenchymal fibrosis, with or without pleural thickening. To diagnose this disorder, one must establish the presence of pulmonary fibrosis and determine whether an exposure has occurred that is of sufficient duration, latency, and intensity to be causal.

Asbestosis becomes evident only after an appreciable latency period, often two decades under current conditions in the United States. In one study of former workers from an amosite asbestos insulation factory that had high levels of asbestos dust, employment for as little as 1 month resulted in a prevalence of 20% of parenchymal opacities 20 years after exposure ceased (70). The duration and intensity of exposure probably influence the length of the latency period: relatively short-term, high-intensity exposures may be associated with a shorter latency than prolonged, lower intensity exposures.

Asbestosis is usually associated with dyspnea, bibasilar rales, and changes in pulmonary function: a restrictive pattern, mixed restrictive–obstructive pattern, and/or decreased diffusing capacity. The abnormal PA chest film and its interpretation remain the most important factors in establishing the presence of pulmonary fibrosis (Figure 8). Compensation systems may require that the chest radiographs be classified by the ILO system once it is established that the patient has been exposed to asbestos. A profusion of irregular opacities at the level of 1/0 is used as the boundary between normal and abnormal in the evaluation of the film, although the measure of profusion is continuous and there is no clear demarcation between 0/1 and 1/0 (Figure 9). When radiographic or lung function abnormalities are indeterminate, HRCT scanning is often useful in revealing characteristic parenchymal abnormalities as well as correlative pleural changes that are highly suggestive of asbestos exposure, particularly when they are bilateral. The specificity of the diagnosis of asbestosis increases with the number of consistent findings on chest film, the number of clinical features present (e.g., symptoms, signs, and pulmonary function changes), and the significance and strength of the history of exposure.

Although asbestosis is characteristically most advanced and appears earliest in the lower lung fields, there is a rare but well-characterized syndrome of massive bilateral upper lobe fibrosis, in the absence of tuberculosis or lung cancer (71–73).

The characteristic change in pulmonary function observed in asbestosis is a restrictive impairment, characterized by reduction in lung volumes (especially the FVC and total lung capacity), decreased diffusing capacity, and arterial hypoxemia (74, 75). Large airway function, as reflected by the FEV_1/FVC ratio, is generally well preserved. In one of the earliest studies conducted, about 50% of asbestos workers presented with FVC below 80% predicted. The frequency of abnormal vital capacity increased, and the mean vital capacity decreased by 18% over the subsequent 10 years (33, 75). The frequency and magnitude of the restrictive defect increased with ILO category (i.e., increased profusion of irregular opacities) and the presence of pleural changes.

Notwithstanding the predominantly parenchymal and restrictive pattern of the disease, airway obstruction can also be observed and can be seen alone in nonsmokers who have asbestosis. These patients usually have a restrictive pattern of lung function, but clinically they also feature an obstructive component charac-

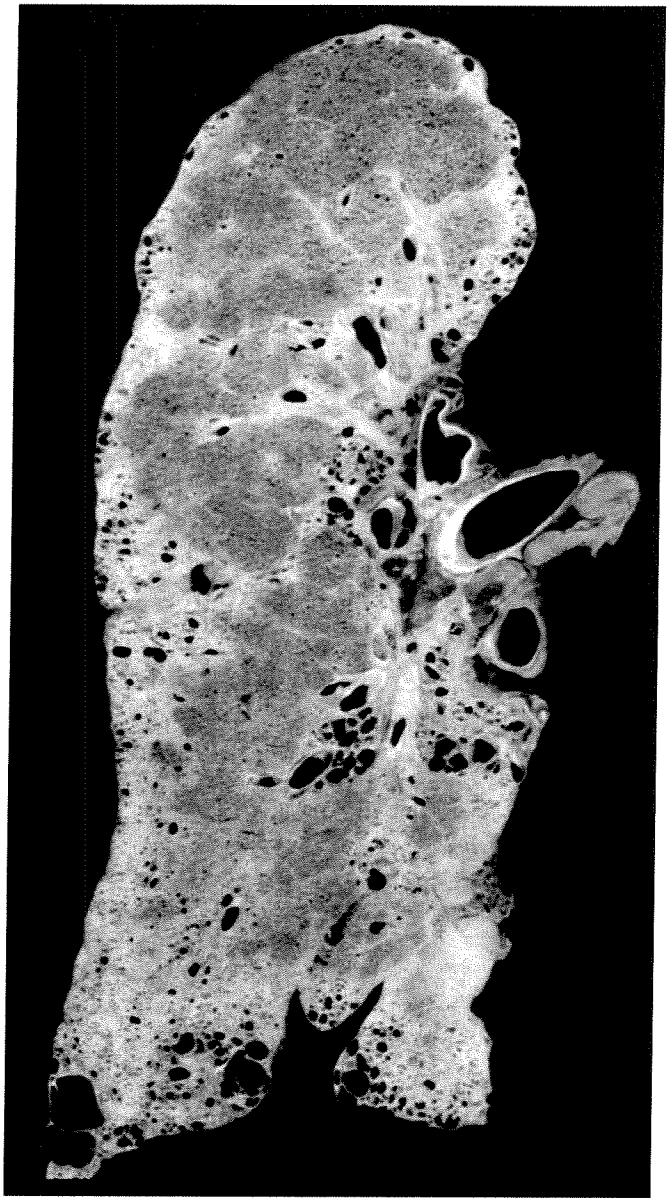


Figure 7. Whole lung section of freeze-dried lung from a person who died of asbestosis. Note the peripheral honeycombing, which is most severe in the lower zones.

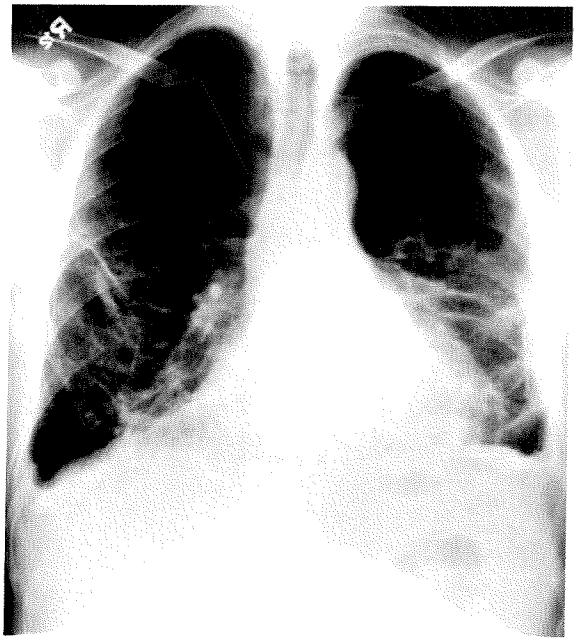


Figure 8. Advanced asbestosis (details of case not available). Note characteristic features: fibrotic bands superimposed on a background of widespread irregular opacities, shaggy heart border and septal thickening, extensive pleural changes, and blunted costophrenic angles.

terized physiologically by increased isoflow volume, and increased upstream resistance at low lung volumes (14, 16). These obstructive findings may be due to asbestos-induced small airway disease. Thus, mixed restrictive and obstructive abnormalities do not rule out asbestosis or necessarily imply that asbestos has not caused an obstructive functional impairment (76).

Asbestosis may remain static or progress; regression is rare (77). The factors that determine prognosis and evolution of the disease are poorly understood. Progression, after cessation of exposure or reduction to current permissible exposure levels, is considerably more common in persons who already have radiographic abnormalities and appears to be associated with level and duration of exposure and therefore cumulative exposure (78).

Differential diagnosis. Although not usually necessary for the diagnosis of asbestosis when a significant exposure history is obtained, lung biopsy may be warranted to exclude other, potentially treatable diseases. Biopsy material may be helpful in identifying the nature of a disease in an indeterminate case or one lacking an adequate exposure history.

The presence of asbestos bodies in tissue sections should be

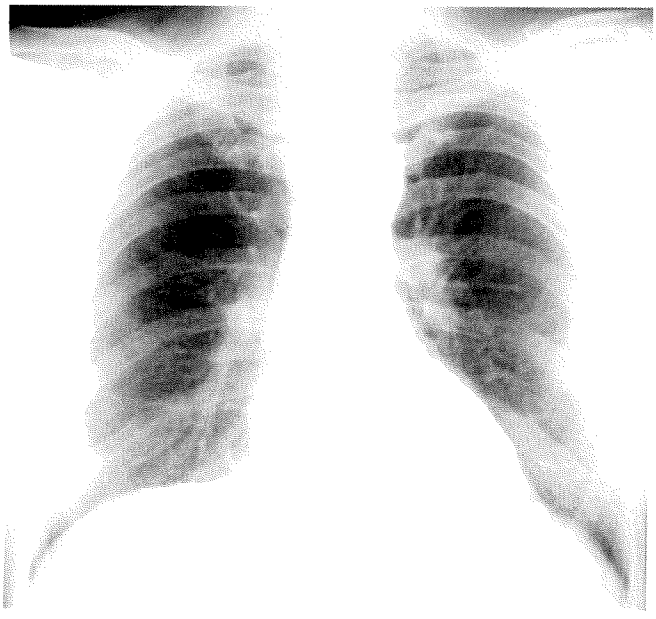


Figure 9. Early asbestosis, showing irregular opacities in lower lung fields that may be categorized as 0/1 or approaching 1/0 according to the ILO classification. Note pleural changes.

sufficient to differentiate asbestosis from other forms of interstitial fibrosis. The chance of finding one asbestos body from background exposure alone has been shown to be about 1 per 1,000 (79). Conversely, the presence of interstitial fibrosis in the absence of asbestos bodies is most likely not asbestosis, although rare cases of pulmonary fibrosis with large numbers of uncoated asbestos fibers have been described (80–82). Idiopathic pulmonary fibrosis (IPF in clinical terms or usual interstitial pneumonitis in terms of pathology) has an acinar pattern of fibrosis different from that of asbestosis and is not associated with asbestos bodies in tissue sections. On occasion, asbestosis is seen in conjunction with an unrelated interstitial lung disease (such as sarcoidosis) or in association with another pneumoconiosis, for example, silicosis. In the absence of fibrosis, asbestos bodies are an indication of exposure, not disease.

Asbestosis resembles a variety of other diffuse interstitial inflammatory and fibrotic processes in the lung and must be distinguished from other pneumoconioses, IPF, hypersensitivity pneumonitis, sarcoidosis, and other diseases of this class. The clinical features of asbestosis, although characteristic, are not individually unique or pathognomonic, but the characteristic signs of the disease are highly suggestive when they occur together. The presence of pleural plaques provides useful corollary evidence that the parenchymal process is asbestos related.

Diagnostic uncertainty is most likely in certain groups of patients. Patients may have a heavy cigarette-smoking history and concurrent emphysema (which also reduces the diffusing capacity). In such cases, one expects a history of asbestos exposure commensurate with the degree of disease. On occasion, a patient with another interstitial lung disease, such as IPF, will have a history of asbestos exposure. Rapid progression, with a visible, year-to-year increase in symptoms, progression of radiographic findings, and loss of pulmonary function in the absence of intense asbestos exposure, suggests the diagnosis of IPF rather than asbestosis.

Patients may be exposed at various times in their working life to more than one dust, such as silica and asbestos, or to mixed exposures, such as dusts in combination with fumes and vapors in welding (83). These patients may have combined disease or the effects of one dust or other exposure may dominate. For example, predominantly upper lobe rounded opacities, hilar node enlargement, and progressive massive fibrosis are not features of asbestosis and if present suggest other causes for the lung disease than asbestos, such as silicosis.

On occasion, isolated fibrotic lesions associated with asbestos resemble solitary pulmonary nodules. These are sometimes called "asbestomas" and usually occur against a background of irregular opacities; they rarely appear in isolation. They normally require biopsy because they are not distinguishable from lung malignancies otherwise (84).

Nonmalignant Pleural Abnormalities Associated with Asbestos

Pleural abnormalities associated with asbestos exposure are the result of collagen deposition resulting in subpleural thickening, which may subsequently calcify, and which in the visceral pleura may be associated with parenchymal fibrosis in adjacent subpleural alveoli (Figures 10 and 11). Pleural thickening, as a marker of asbestos exposure, has continued to be a prominent feature of exposure to asbestos while other outcomes, such as asbestosis, have become less frequent due to declining exposure levels. The major determinant of pleural thickening is duration from first exposure (70).

It is unclear whether the relative frequency of diffuse and circumscribed pleural thickening has changed. The *International Classification of Radiographs of Pneumoconioses* (38) provides a basis for recording and classifying both types of pleural thickening, allowing correlation with indices of exposure and measurements of lung function. Manifestations of disease of the lung and of the pleura have become less evident and less characteristic on plain films as exposures have decreased. However, CT scan (including high-resolution images) detects pleural thickening not evident on the plain film, and sometimes fails to confirm apparent pleural thickening read on the plain film. Schemes to quantify extent of pleural thickening on CT scan have been published (55, 85). Rarely, interlobar pleural thickening may mimic lung nodules on CT scan (86).

Pleuritis: acute pleural effusion, chronic pleuritic pain. Asbestos may cause an acute pleural effusion, often lasting several months, that is exudative and often hemorrhagic, with variable numbers of erythrocytes, neutrophils, lymphocytes, mesothelial cells, and often eosinophils (87–89). It may occur early (within 10 years, unlike other asbestos-related diseases) or late after the onset of asbestos exposure (90). It may be superimposed on long-standing pleural plaques (91). Although it is usually asymptomatic, the acute pleural effusion due to asbestos may also be exuberant, with fever and severe pleuritic pain. It is sometimes detected only incidentally on a radiograph taken for another purpose (87, 88). The effusion may persist for months, present bilaterally, or recur on the same or the opposite side (87). A friction rub may be present (92, 93). The traces of pleural effusion may be observed years later as a blunted costophrenic angle or as diffuse pleural thickening. Acute pleuritis is thought to underlie many cases of diffuse pleural thickening. Of 20 insulators with a past history of definite pleural effusion, diffuse pleural thickening was detected on radiograph in 16 (90). Dose–response relationships or characteristic features of exposure associated with effusion have not been described.

Chronic severe pleuritic pain is rare in patients with asbestos-related pleural disease (92, 93). Vague discomfort appears to be more frequent. Studies examining the frequency of atypical chest pain in asbestos-exposed patients have not been performed. In the few cases described, it was present for many years, disabling, and often bilateral. Radiographic evidence of pleural disease ranged from plaques to extensive diffuse and circumscribed pleural thickening; several cases followed pleural effusions. The diagnosis of acute asbestos-related pleural effusion is by exclusion of other causes of acute pleuritis, and most often is not arrived at until the pleural space is fully explored and biopsied, generally by thoracoscopy. Differentiation from Dressler's syndrome is difficult in asbestos-exposed patients who have undergone recent cardiac surgery. Differentiation from mesothelioma or pleural extension of a pulmonary malignancy is critical, and may be difficult on clinical grounds (including positive gallium and positron emission scan). Pleural fluid cytology is useful for distinguishing benign from malignant effusions. It is not unusual for nonspecific effusions to precede mesothelioma by several years. If a malignancy has not manifested itself within 3 years, the effusion is generally considered benign.

The diagnosis of chronic pleuritis manifested by pleuritic pain is reached by excluding malignancies, because most other causes of acute pleuritis do not result in chronic pain. Malignancy is unlikely when pain persists for years with little or no clinical or radiographic change.

Plaques: circumscribed pleural thickening. Pleural plaques are indicators of exposure to asbestos. They are clearly the most common manifestation of the inhalation, retention, and biologic effect of asbestos. Their prevalence is most directly related to duration from first exposure; they are rare within less than 20 years. Pleural plaques consistent with asbestos exposure appear in chest films of 2.3% of U.S. males, a percentage that has been

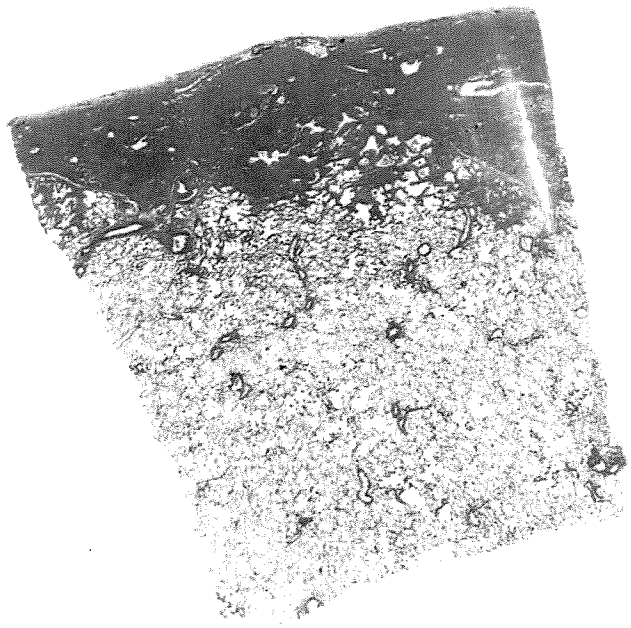


Figure 10. Photomicrograph of H&E-stained section of lung from a person with mild asbestosis. There is marked fibrosis of the pleura with some subpleural fibrosis. Higher power magnification of the same section showed that minimal disease was also present around the small respiratory bronchioles.

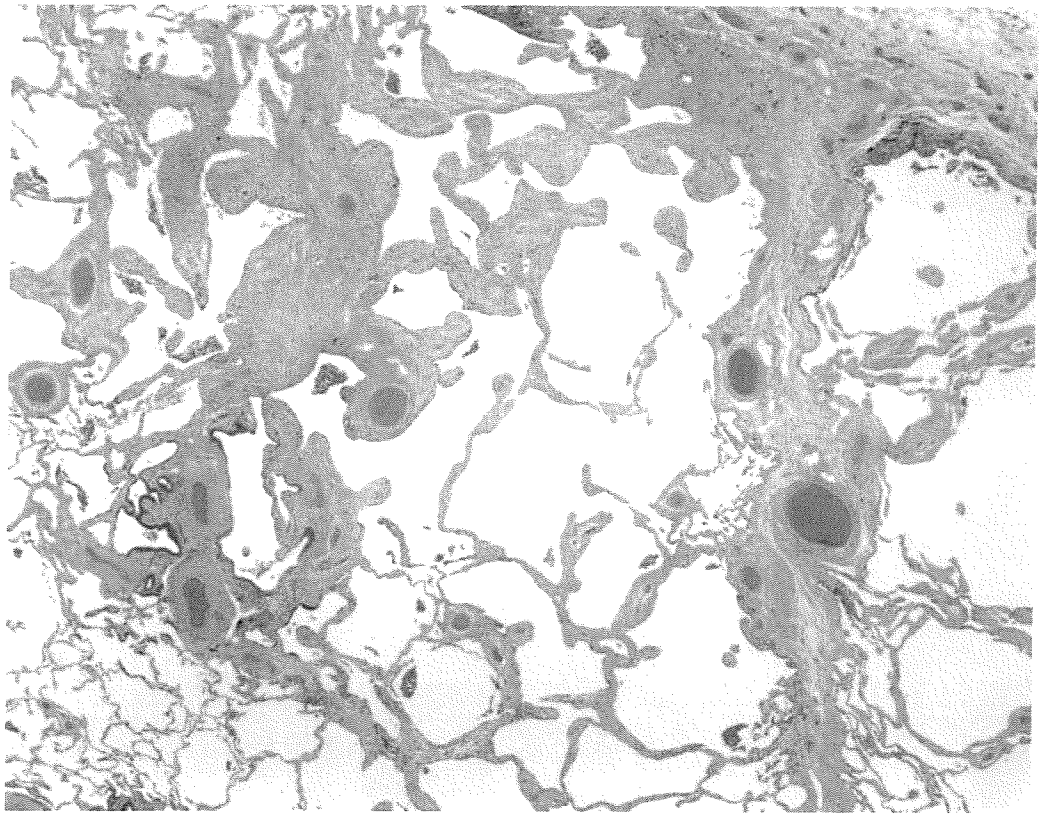


Figure 11. Photomicrograph of H&E-stained section of a person with Grade III asbestosis showing fibrosis in the lung parenchyma and overlying visceral pleura, with extension of the fibrosis into the interlobular septa.

Figure 12. Gross appearance at autopsy of asbestos-associated pleural plaques overlying the lateral thoracic wall.

remarkably stable both for the general population in the early 1970s and veterans in the 1990s (94, 95).

Calcification is similarly related to duration. Smoking plays no role in the prevalence of pleural plaques (68). Pleural plaques are bilateral, but not symmetric, lesions of the parietal pleura. Characteristically, they are found following the ribs on the lower posterior thoracic wall (Figure 12) and over the central tendons of the diaphragm (Figure 13). They are raised, sharply circumscribed with a smooth or with a rounded knobby surface, and range in color from white to pale yellow. They generally spare the costophrenic angles and apices of the thoracic cavity. Microscopically, they consist of mature collagen fibers arranged in an open basket-weave pattern and are covered by flattened or cuboidal mesothelial cells. They are relatively avascular and acellular and show minimal inflammation. They are sharply demarcated from subpleural tissues and central calcification is common. Asbestos bodies are not seen in or adjacent to the lesions (64). Isolated plaques may be associated with tuberculosis, trauma, and hemothorax; however, multiple lesions having the classic appearances described above are almost invariably associated with asbestos exposure.

The conventional chest film is a sensitive and appropriate imaging method for plaques, although it may identify abnormalities that resemble plaques but are not. In the PA radiograph, they are best seen in profile on the midlateral chest walls and on the diaphragm or face on, and show serrated borders. HRCT is not a practical screening method for demonstrating plaques because of the separation between sections, the high radiation exposure, and the lack of access to the test in some locations. HRCT is useful to identify questionable abnormalities and to resolve questions about structures that resemble plaques.

Typical pleural plaques are easily identified on plain films by sharp, often foliate, borders (face on) and by a raised straight surface with clear, cut-off edges when seen face on (Figures 14–16) and as irregular margins (sometimes almost rectangular) when seen in profile on the chest wall or diaphragm. Apparent pleural thickening with gradually tapering or indistinct edges is often due to subpleural fat or superimposed soft tissue; fat pads below the parietal pleura typically occur in the midthoracic wall,

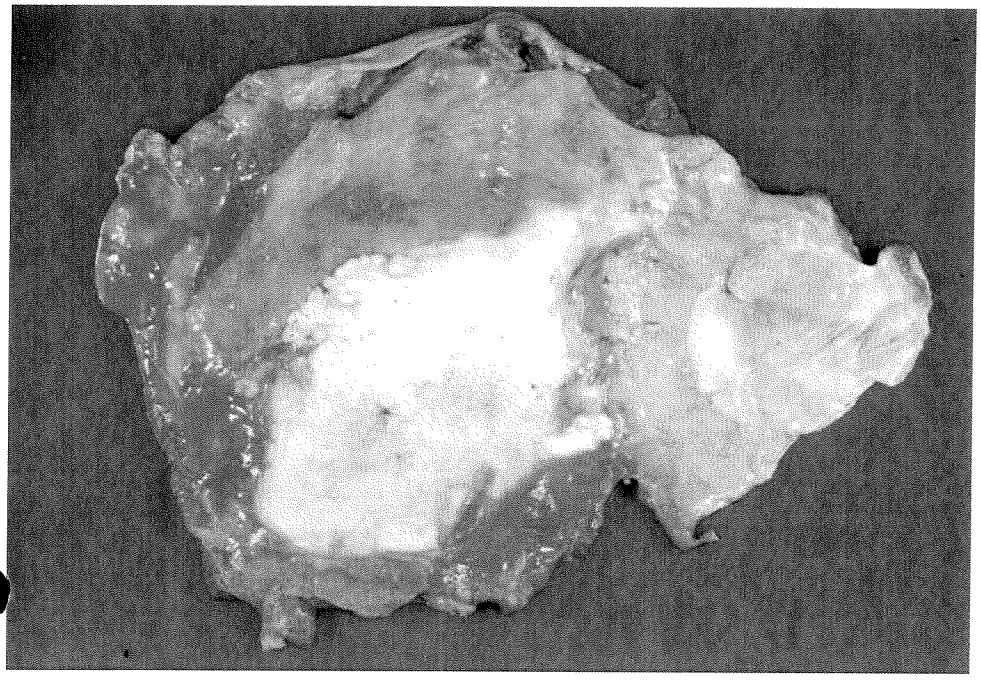


Figure 13. Gross appearance of large asbestos-related pleural plaque over the dome of the diaphragm.

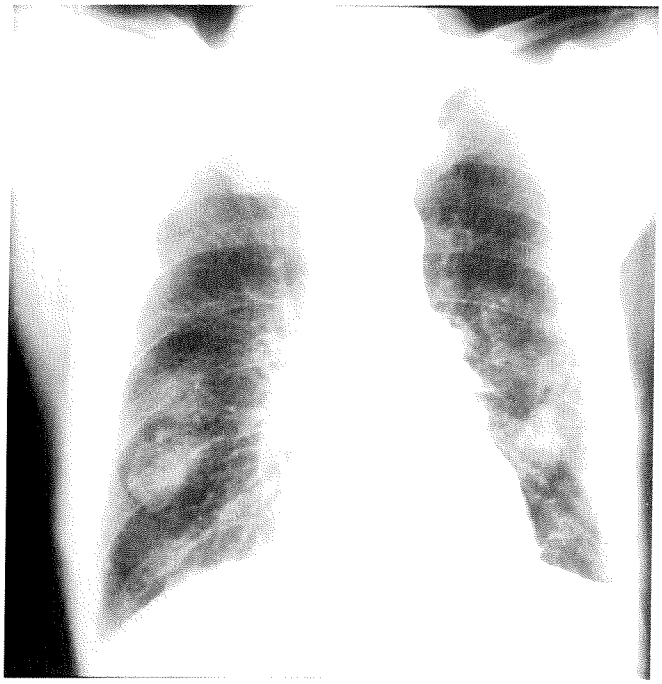


Figure 14. En face (face on) pleural plaques in a chest film with minimal parenchymal disease; worker was 54 years old at the time this chest film was taken (1982) and was exposed to asbestos in the 1960s as an insulation worker.

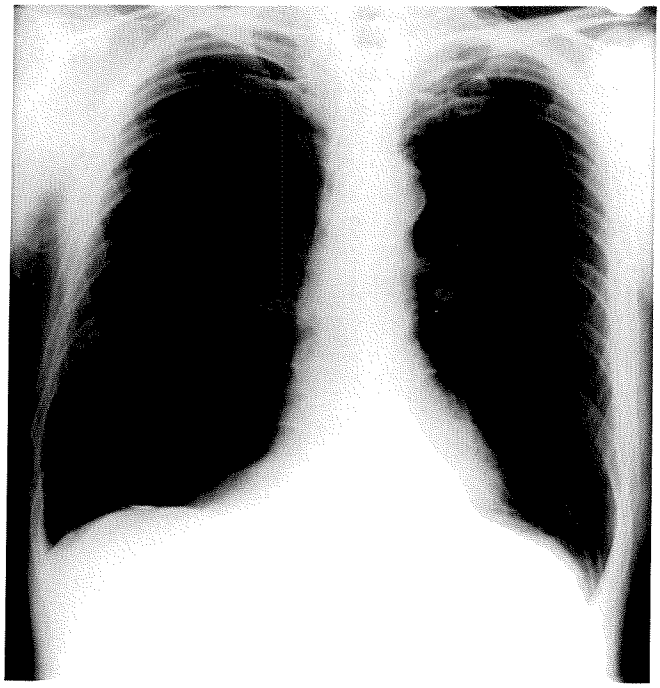


Figure 15. Pleural plaque, with linear calcification, seen on edge on the right hemidiaphragm in a 72-year-old sheet metal worker. No visible parenchymal disease.

between the fourth and eighth ribs, as do pleural plaques (51). Proper penetration is important on plain film; differentiation of fat from pleural plaques may still be difficult but is readily made by HRCT. Less typical plaques on the diaphragm may be difficult to detect and should be distinguished from atelectatic streaks, visceral folds, or diaphragmatic straightening caused by bullae. Calcification is helpful but may not be apparent in an underpenetrated film (Figure 14). Axial CT scans often fail to image diaphragmatic plaques (96).

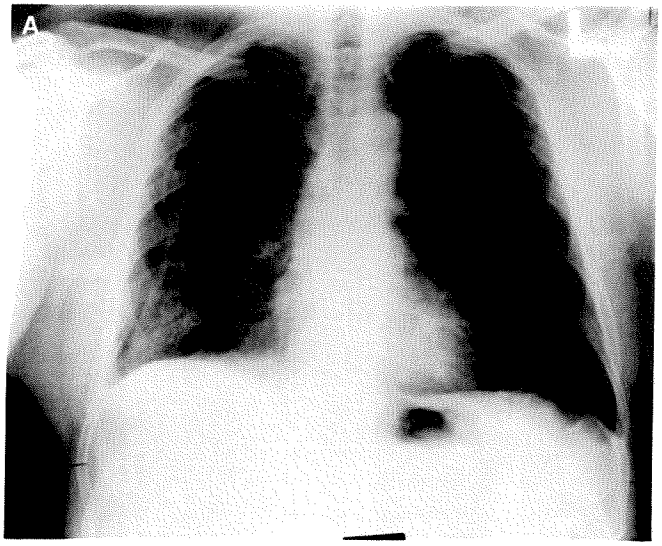
The origin of pleural plaques is not clear (97, 98). The burden of asbestos fibers in lung tissue and of asbestos bodies in bronchoalveolar lavage fluid is greatly increased in patients with diffuse pleural thickening or asbestosis and moderately increased in patients with pleural plaques compared with unexposed subjects (99–101). The presence of pleural plaques is correlated with parenchymal disease, in particular fibrotic bands and both peribronchiolar and alveolar fibrosis. However, peribronchiolar fibrosis is absent in many cases with pleural plaques and present in many cases without them (102).

Slow progression of plaques is typical. Approximately 85% of heavily exposed workers showed pleural thickening (predominantly plaques) on plain film more than 40 years from first exposure (103), as did up to 17% of environmentally exposed populations (104). More than half the cases were bilateral.

The presence of plaques is associated with a greater risk of mesothelioma and of lung cancer compared with subjects with comparable histories of asbestos exposure who do not have plaques (105, 106). This is thought to be due to greater exposure or retained body burden, not malignant degeneration. Therefore, the presence of pleural plaques should be interpreted as a marker for elevated risk of malignancy, which may be higher than the occupational history alone might suggest.

Although pleural plaques have long been considered inconsequential markers of asbestos exposure, studies of large cohorts have shown a significant reduction in lung function attributable to the plaques, averaging about 5% of FVC, even when interstitial fibrosis (asbestosis) is absent radiographically (74, 76, 107). The presence of circumscribed plaques can be associated with restrictive impairment and diminished diffusing capacity on pulmonary function testing, even in the absence of radiographic evidence of interstitial fibrosis (108, 109). Taking into account the degree of interstitial fibrosis as measured by ILO profusion score (described below), smoking, and duration of asbestos exposure, significant decrements in vital capacity have been observed: a reduction of up 140 ml or more of FVC associated with circumscribed plaques (76). This has not been a consistent finding (110, 111) and longitudinal studies have not shown a more rapid decrement in pulmonary function in subjects with pleural plaques (112). Decrements, when they occur, are probably related to early subclinical fibrosis. Dyspnea on exertion was reported more often among subjects with circumscribed pleural thickening independent of parenchymal disease and appeared to be proportional to the extent (110). There is a significant but small association between the extent of circumscribed pleural plaques and FVC, which is not seen with diffuse pleural thickening (112, 113). Even so, most people with pleural plaques alone have well preserved lung function (55).

It is unclear whether this small effect on lung function is sufficient to contribute to dyspnea but there is evidence that it might. Half of subjects with pleural thickening but normal chest films and normal lung function showed excessive ventilation with exercise, which can contribute to dyspnea (114). Excessive ventilation on exercise could be the result of decreased chest wall and/or lung compliance caused by pleural thickening alone or to decreased lung compliance and ventilation–perfusion imbalance caused by parenchymal fibrosis that was not detected radiographically.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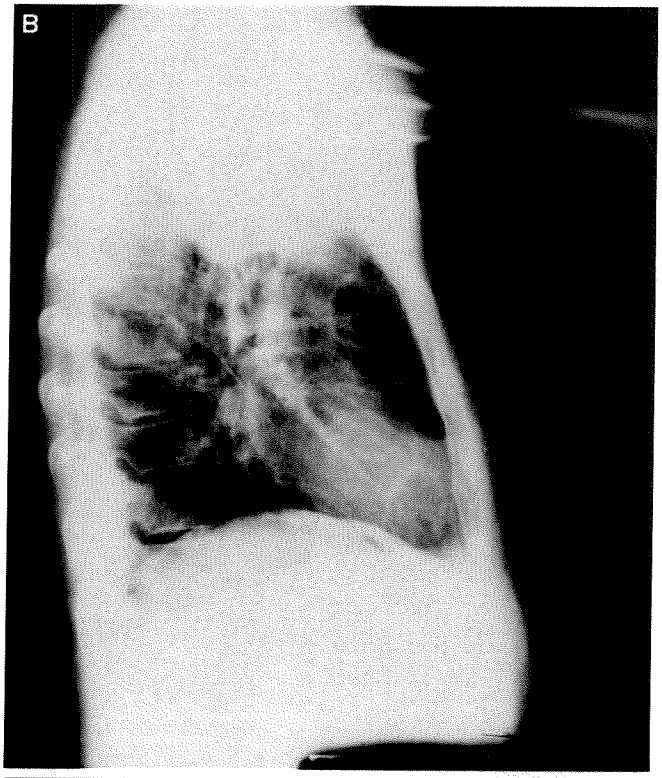
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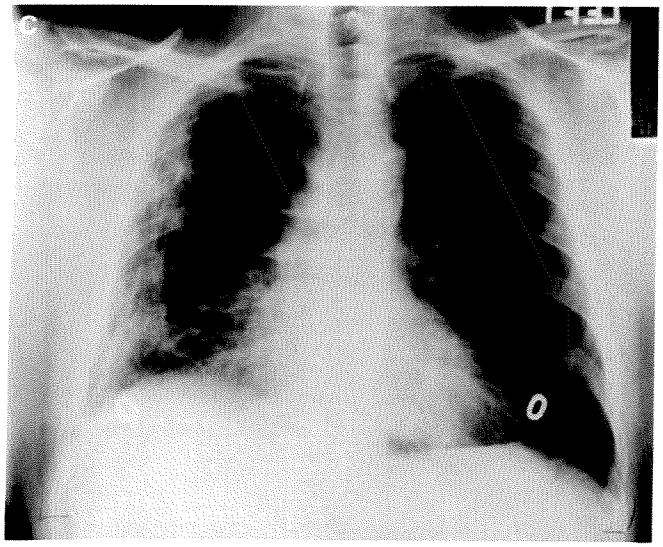
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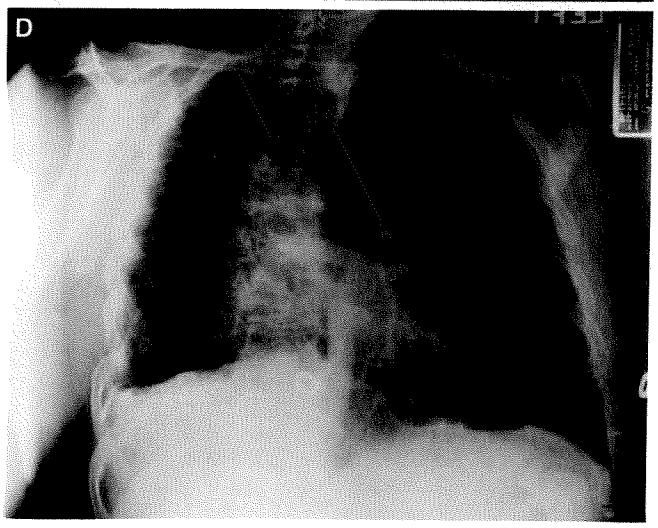
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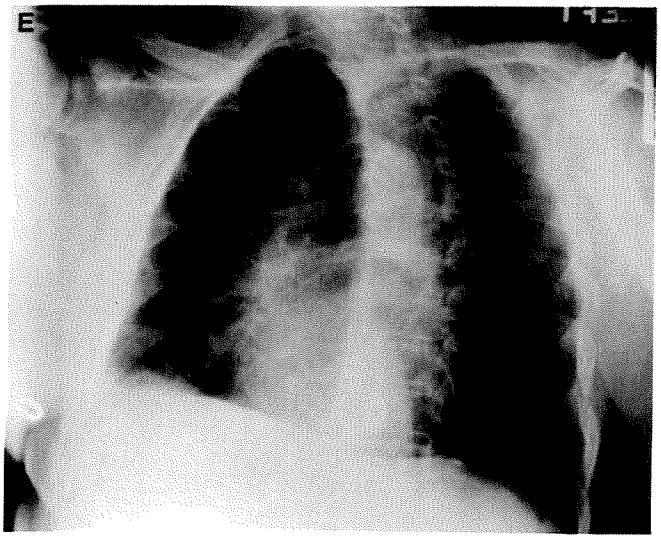
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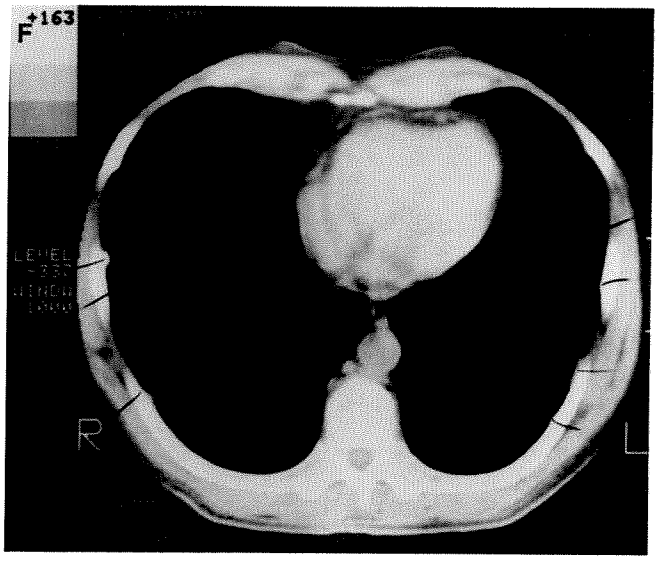
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Plaques are indicators of increased risk for the future development of asbestosis (94). This may reflect greater exposure or retained body burden. An autopsy study has demonstrated more frequent peribronchiolar fibrosis when plaques are present (90). This finding, as well as derangements in gas exchange (114) and evidence from HRCT, indicate that subradiographic asbestosis may be present in some patients with only pleural plaques. The presence of plaques is therefore an indication to monitor the patient over time for interstitial fibrosis (115).

Diffuse pleural thickening. Diffuse thickening of the visceral pleura is not sharply demarcated and is often associated with fibrous strands ("crow's feet") extending into the parenchyma. In large surveys of asbestos-exposed workers, diffuse pleural thickening has ranged from 9 to 22% of those with pleural disease. Both circumscribed and diffuse pleural thickening may be present in the same hemithorax. Diffuse pleural thickening superimposed on circumscribed plaques has been observed, often after pleural effusion (91).

The frequency of diffuse pleural thickening increases with time from first exposure and is thought to be dose related (104). Diffuse pleural thickening has been observed after acute pleuritis (90). It may also be caused by extension of interstitial fibrosis to the visceral pleura, consistent with the pleural migration of asbestos fibers. The extent of diffuse pleural thickening seems to be more or less uniformly distributed, the different degrees being fairly equally often seen, however, in contradistinction to circumscribed pleural thickening, in which the lowest categories are more frequent (113). Lung burdens of asbestos in these cases are intermediate between asbestosis and pleural plaques (116–118).

This condition affects the visceral pleural surface and is quite different in appearance from the parietal pleural plaque. It consists of pale gray diffuse thickening that blends at the edges with the more normal pleura. It may be extensive and cover a whole lobe or whole lung and obliterate lobar fissures. It ranges in thickness from less than 1 mm up to 1 cm or more. Adhesions to the parietal pleura are common, particularly opposite to pleural plaques. The lesion may show a gradient with immature granulation tissue and fibrin at the surface, progressing to mature collagen adjacent to the lung. The fibrosis may extend for a few millimeters into the lung parenchyma and into the lobular septae. The latter features do not constitute asbestosis.

Diffuse pleural thickening may have a significantly greater impact on pulmonary function than circumscribed plaques. A reduction of 270 ml of FVC has been associated with diffuse pleural thickening (76, 119). Workers with diffuse pleural thickening have a significantly greater decrement in FVC (by a factor of two or more) than those with circumscribed pleural thickening (76, 113). This effect is unrelated to the radiographic extent of pleural thickening; a similar reduction in FVC was seen with little more than costophrenic angle blunting as with extensive involvement (113). Decrements associated with diffuse pleural thickening reflect pulmonary restriction as a result of adhesions of the parietal with the visceral pleura. Restrictive impairment is characteristic, with relative preservation of diffusing capacity (pattern of entrapped lung).

Diffuse pleural fibrosis extends continuously over a portion of the visceral pleura, often causing adhesions to the parietal pleura, involving the fissures and obliterating the costophrenic angle. The newly revised ILO classification (2003) recognizes pleural thickening as diffuse "only in the presence of and in continuity with, an obliterated costophrenic angle" (38). Localized subpleural parenchymal fibrosis is often present without diffuse interstitial fibrosis (117). Calcification of the pleura occurs with the passage of time, and may involve fissures. A rare variant of visceral pleural fibrosis is progressive apical thickening associated with fibrosis of the upper lobe (120, 121).

Pachypleuritis is extensive, often bilateral, pleural fibrosis with evidence of active inflammation histologically and by gallium uptake. Extension of fibrosis into the lung is often evident radiographically as irregular pleural and pericardial borders, fibrous streaks, or "crow's feet" and bands. Ventilatory failure leading to CO_2 retention, cor pulmonale, and death has been described in four patients with bilateral involvement and little or no parenchymal fibrosis, and in one patient with unilateral pleural thickening. Decortication may be beneficial (122).

Rounded atelectasis. Rounded atelectasis (123, 124), also known as shrinking pleuritis, contracted pleurisy, pleuroma, Blesovsky's syndrome (125), or folded lung, presents radiographically as a mass and may be mistaken for a tumor (Figure 17). The condition may result from pleuritis of any cause. The lesion is thought to develop from infolding of thickened visceral pleura with collapse of the intervening lung parenchyma. Clinical experience suggests that it is more likely to occur today as a result of asbestos exposure than other causes. The classic "comet sign" is pathognomonic and is often more readily seen on an HRCT than on plain films. Clues to its identity are a band connecting the mass to an area of thickened pleura and a slower evolution than that of a lung cancer, so that previous films will show a similar finding. Histologic examination shows folded and fibrotic visceral pleura with atelectasis and variable amounts of chronic inflammation in the adjacent lung parenchyma. The sudden appearance of rounded atelectasis may follow acute pleuritis with effusion. Rounded atelectasis may be multiple and bilateral (124, 126).

Rounded atelectasis is important for the diagnostic pathologist to recognize as it is frequently removed surgically as a suspected peripheral lung cancer. Asbestos bodies and/or evidence of asbestosis should be carefully sought.

Differential diagnosis, including rounded atelectasis and apical thickening. Acute pleuritis of any cause can result in diffuse pleural thickening that is indistinguishable from that associated with asbestos, although such causes are usually unilateral. The most likely causes, empyema, tuberculosis, and trauma, including surgery, are likely to be identified in the medical history. Empyema in childhood or an infected pleural effusion associated with pneumonia may not be.

The major differential diagnostic consideration with diffuse pleural thickening is mesothelioma, which is progressive and more likely to be symptomatic at the time of detection. On occasion, when fibrosis and mesothelial proliferation are exuberant, the distinction is difficult clinically, radiographically, and histologically. Apical thickening (120, 122) must also be distin-

Figure 16. Extensive evaluation in 1983 of a 65-year-old business executive who, in the 1950s, had worked in shipyards for approximately 2 years and was exposed to high levels of asbestos. This case is unusual because both early asbestosis and a huge pleural plaque are unilateral. (*A*) PA film shows asbestosis and an extensive pleural plaque extending over three-quarters of the length of the hemothorax. Right costophrenic angle is blunted but would not satisfy strict criteria for this according to the ILO classification. (*B*) Lateral film, showing extensive calcified plaques over diaphragm, also visible on *left* in PA film. (*C*) Because of concern for possible mass in right lower lung lobe, PA film was repeated with nipple markers: mass not seen in this view. (*D*) Left anterior oblique, showing absence of other plaques on chest wall. (*E*) Right anterior oblique, showing detail of plaque. (*F*) CT scan, showing plaque.

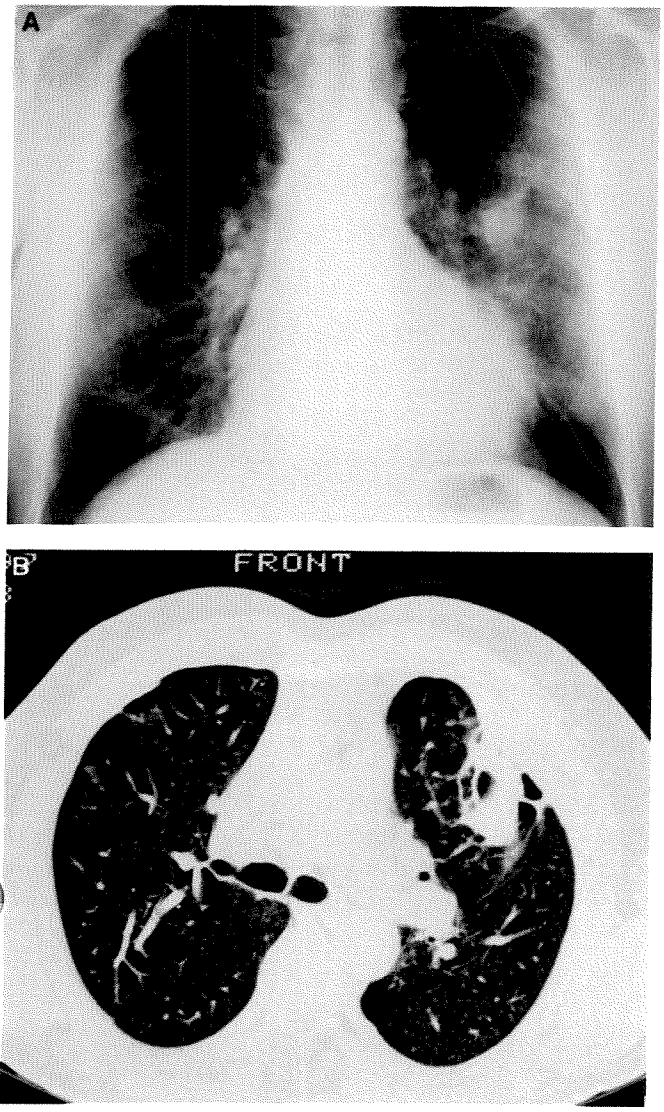

Figure 17. Rounded atelectasis in a 57-year-old sheet metal worker. (*A*) Presentation as a mass in the left chest. (*B*) CT scan showing pleural base and infolding of structures.

guished from mesothelioma and tuberculosis, which may be suggested by history and (previous) bacteriologic findings.

Chronic Airway Obstruction

Asbestos exposure has traditionally been considered to cause predominantly restrictive physiologic abnormalities. The role of asbestos as a cause of airway obstruction has been controversial. However, asbestos exposure has long been known to be associated with an obstructive physiological abnormality (127–129). This association might arise in one or more of several ways:

- Asbestos specifically causes obstructive abnormality.
- Asbestos causes obstructive abnormality nonspecifically (i.e., as do large burdens of most inorganic dusts) (83, 130).
- Work leading to extensive asbestos exposure is frequently associated with exposure to other agents affecting airways.
- Confounding by tobacco smoking may lead to an association.
- Anatomic and physiologic airway abnormalities develop as part of the pathophysiologic process of asbestosis and are not an independent entity.

Asbestos-related chronic airway obstruction may result in reduction in the FEV_1/FVC ratio associated with reduced FEV_1 (29, 76, 113, 127). Epidemiologic studies have demonstrated a significant association between asbestos exposure or asbestosis category as defined radiographically and reduction in FEV_1, FEV_1/FVC ratio, and midexpiratory flow rates (111, 130–133). The relationship between surrogate measures of exposure and the FEV_1 and FEV_1/FVC ratio also occurs in subjects who do not have radiographic evidence of asbestosis (defined as an ILO score exceeding 1/0) (130, 133, 134). A small effect has been observed in lifelong nonsmokers (14, 113, 135, 136). This effect begins in small airways, consistent with the known pathology of bronchiolitis in early asbestosis (136, 137). Radiographically, airflow abnormalities may also be associated with emphysema (138).

Histologically, inflammation and airway fibrosis characterize asbestos-related small airway disease. A major site of asbestos deposition is in the walls of membranous and respiratory bronchioles. In the walls of membranous bronchioles this leads to fibrosis and smooth muscle hyperplasia that are similar, but more severe, than that produced by cigarette smoking (128, 139) (Figures 4, 5, and 18). The respiratory bronchioles show fibrosis, which extends into the alveolated portions of the walls and alveolar ducts (Figure 19). In this regard, it differs from the lesion of cigarette smoking, which primarily involves the nonalveolated portions of the first generation of respiratory bronchioles (140). Asbestos bodies are not present in the walls of the membranous bronchioles, although inflammatory changes are present, but are commonly seen in the walls of the respiratory bronchioles and/or adjacent alveoli. Some authorities consider it appropriate to describe these lesions as true asbestosis because the walls of respiratory bronchioles are largely alveolated and therefore within the gas exchange region of the lung (64). Others consider the small airway lesions as distinct from asbestosis and refer to the lesions of both membranous and respiratory bronchioles as asbestos-induced small airway disease (12). These small airway lesions are the likely anatomic basis for airflow limitation in asbestos-exposed individuals.

In general, the magnitude of the asbestos effect on airway function is relatively small. This effect, by itself, is unlikely to result in functional impairment or the usual symptoms and signs of chronic obstructive pulmonary disease. However, if superimposed on another disease process, the additional loss of function due to the asbestos effect might contribute significantly to increased functional impairment, especially in persons with low lung function.

Asbestos exposure independently contributes to accelerated decline in airflow over time, whether or not exposure ceases (77, 129, 133, 134, 141). Dyspnea, cigarette smoking, diffuse pleural thickening, honeycombing observed on HRCT scan, and indicators of active inflammation have been associated with worsening obstruction (142). Effects on measures of early small airway dysfunction (e.g., midexpiratory flow rates) in themselves are unlikely to produce clinically relevant impairment, but may indicate an increased probability that disease will develop later (128, 129, 134, 143). Development or persistence of respiratory symptoms among asbestos-exposed workers is associated with accelerated loss of lung function, both FVC and FEV_1 (30). In patients with severe obstructive airway disease from another cause, the additional contribution of asbestos-related airflow obstruction might be functionally significant at low levels of lung function. Short duration and low cumulative exposure are less likely to produce significant obstructive abnormality (112, 134).

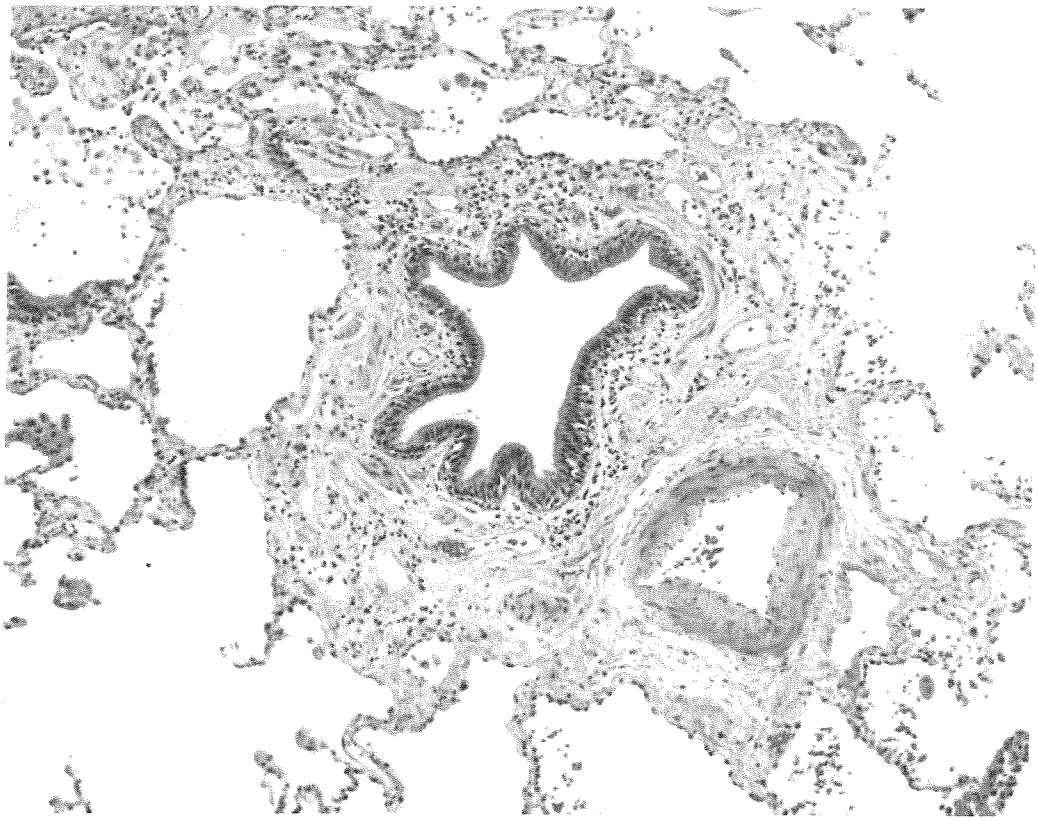

Figure 18. Photomicrograph of asbestos-related small airway disease, showing thickened membranous bronchiole. There is also fibrosis around the airway, and a mild chronic inflammatory cell infiltrate in its wall.

Assessment of functional impairment of clinical significance (3) should generally be based on the restrictive findings associated with asbestosis, as these are more likely to be disabling. However, the addition of obstructive disease adds to the level of functional impairment (144). Treating restriction and obstruction separately may underestimate their combined effect on impairment. The normal indicator for restrictive impairment, total lung capacity, has proven to be insensitive to total impairment in subjects with both asbestosis and chronic obstructive lung disease. In such cases, diffusing capacity and alveolar–arterial oxygen difference may be more revealing (144). Some of the restrictive component may be contributed by air trapping rather than fibrosis (145).

Chronic obstructive airway disease that is not due to asbestos (e.g., secondary to smoking) may complicate the recognition of asbestosis. For example, total lung capacity may be normal when both disorders are present, due to a restrictive process offsetting air trapping (143). Whereas the FEV_1/FVC ratio may be reduced in asbestos-exposed persons with no or a low profusion of small, irregular opacities, this ratio may also be normal in more advanced asbestosis (i.e., with higher profusion and diminished FVC) because of a reduction in FVC (75).

Effects on airflow begin before the development of asbestosis (129). In individuals who develop asbestosis, physiologic findings associated with airflow obstruction (e.g., reduction in the FEV_1/FVC ratio) become less prominent as asbestosis progresses; this may reflect increased pulmonary recoil.

The dose and time course of asbestos-associated airway abnormalities have received limited attention. Many available stud-

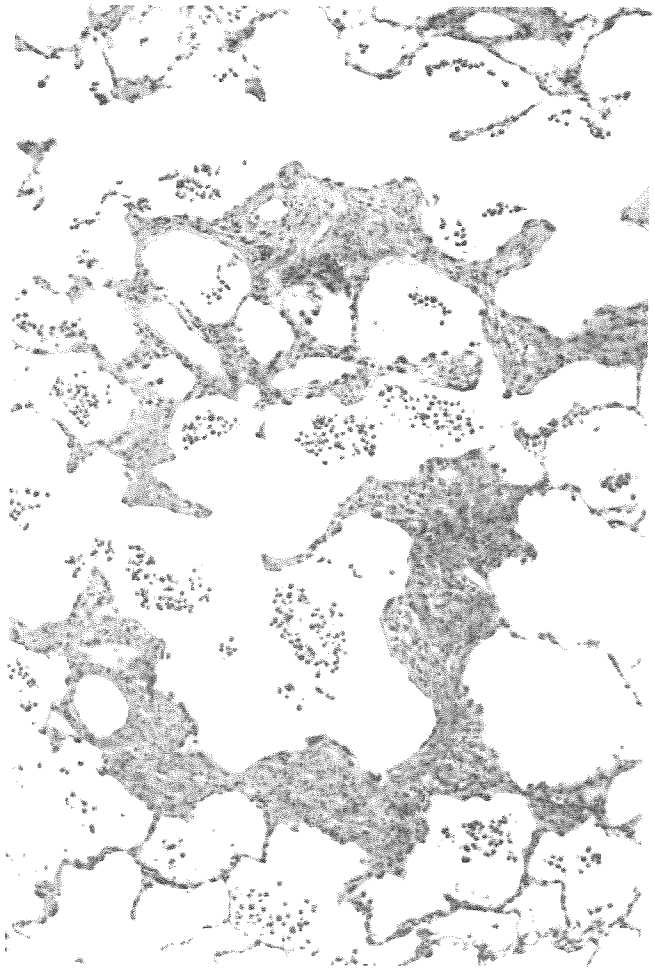

Figure 19. Photomicrograph of asbestos-related small airway disease, in this case a respiratory bronchiole, with extension of the fibrosis into the adjacent parenchyma (Grade II asbestosis; *see* Table 2).

TABLE 3. RECOMMENDATIONS FOR MANAGEMENT AFTER DIAGNOSIS OF ASBESTOSIS

1. Patient notification
   1.1. Inform patient of work-related illness
   1.2. Report to appropriate authority as occupational disease, as required by law
   1.3. Inform patient that there are options for compensation
2. Impairment assessment
   2.1. Conduct an assessment of functional impairment
   2.2. Rate impairment in accordance with ATS criteria,* which are incorporated into the AMA Guides†
3. Tertiary prevention
   3.1. Smoking cessation (primary prevention for smoking-related disorders)
   3.2. Withdrawal from further excessive exposure‡
   3.3. Immunization (pneumococcal pneumonia, influenza)
   3.4. Management of concurrent respiratory and other diseases
4. Monitoring
   4.1. Chest film and pulmonary function testing§ should be conducted every 3 to 5 years
   4.2. Active monitoring (periodic screening) for colon cancer
   4.3. Observation and elevated index of suspicion but not screening for lung cancer, mesothelioma, gastrointestinal cancers (other than colon)
5. Development of a patient-specific management plan for symptomatic disease

Definition of abbreviations: AMA = American Medical Association; ATS = American Thoracic Society.
* *See* Reference 3.
† *See* Reference 157.
‡ *See* text.
§ *See* References 4 and 5.

ies reflect relatively high historical levels of exposure. Among nonsmoking Chinese asbestos workers, association of cumulative exposure with functional effects was seen only among those with long-term exposure (133).

Tobacco smoking is the predominant cause of chronic airway obstruction in asbestos-exposed workers who smoke, although occupational exposures can be significant. The association between airway obstruction and exposure to asbestos has been well demonstrated in nonsmokers, and in some studies the association between exposure and airway obstruction is seen only among nonsmokers (131); among smoking asbestos-exposed workers, smoking accounts for most of the small airway abnormality (111, 127, 135, 141, 142). In addition to smoking, other occupational exposures might contribute to chronic obstructive airway disease; effects of asbestos in producing airflow obstruction are likely to be additive to these. There may be an interaction between smoking and asbestos in the development of airway obstruction, as has been demonstrated in animal models (146), but this has not yet been demonstrated for human subjects.

IMPLICATIONS OF DIAGNOSIS FOR PATIENT MANAGEMENT

A history of significant asbestos exposure obligates the responsible physician to provide a management plan for the patient that takes into consideration current disease and impairment as well as future risk (147). A recommended management plan is summarized in Table 3.

Workers referred for evaluation of asbestos-related disease today differ from those referred in past years. Exposure to asbestos among these workers is likely to be more remote in time and to have been less intense. Exposed workers may live longer and progress later to more advanced stages of disease. They are more likely to survive to develop additional outcomes associated with asbestos, such as malignancy, and to present more complicated management challenges (148).

Actions Required before Disease Is Apparent

A recent or short-term history of exposure to asbestos, particularly in the absence of detail on duration and intensity, requires the clinician at a minimum to educate the patient with respect to latency, the exposure–response relationship characteristic of asbestos-related diseases, and the future risk of malignant disease. Reassurance should be offered where appropriate and the risk placed into the context of the exposure history. This is often an excellent opportunity at the same time to review the patient's history, work hygiene practices, behavior and attitudes toward cigarette smoking, as well as exposure to other occupational and environmental carcinogens (149).

For all patients presenting with a history of significant or possibly significant exposure, at a minimum a baseline, high-quality chest film should be obtained, together with spirometry and a single-breath diffusing capacity that conform to American Thoracic Society guidelines. Complete pulmonary function testing should be obtained if clinically indicated. Workers who have had exposure to asbestos have also often worked in other dusty occupations. They and their families may have lived in communities where they experienced environmental exposures.

The sensitivity of the plain chest film for identifying asbestosis at a profusion level of 1/0 (in the ILO classification system) has been estimated at or slightly below 90%. The corresponding specificity has been estimated at 93%. Applied to populations with varying prevalence of disease, the positive predictive value of the minimally abnormal chest film alone in making the diagnosis of asbestosis may fall below 30% when exposure to asbestos has been infrequent and exceed 50% when it has been prevalent. This suggests that screening programs based on the chest film alone may vary considerably in their yield of true cases depending on the characteristics of the population being screened. In the general population and for occupational groups with low levels of exposure they may be unreliable in identifying asbestosis. The application of multiple criteria, as outlined in this statement, is a preferable approach (150). However, combinations of tests for a specific criterion, such as a hypothetical requirement that multiple tests for pulmonary function be abnormal, would reduce the sensitivity without enhancing specificity for asbestos-related disease; in general, the most sensitive test for a particular criterion is preferable (2).

Persons identified as having asbestos-related disease or a significant exposure history should be informed of the risk of progression of disease, the risk of malignancy, and especially

the interaction between smoking and asbestos exposure in enhancing the risk of lung cancer. Such persons who smoke may be more motivated to consider cessation when the connection between asbestos and the risk of respiratory impairment and of malignancy is brought up at this time (151). The risk conferred by other occupational and environmental carcinogens should also be emphasized at this time.

The question of monitoring for asbestos-related disease is complicated by requirements for occupational surveillance, especially for those with minimal exposure. The Occupational Safety and Health Administration asbestos standard requires employers to monitor their asbestos-exposed workers during employment but makes no provision beyond the period of employment, despite the latency, and private insurance may or may not allow the expense thereafter (8).

Persons with a history of exposure to asbestos but no manifest disease, and for whom the time since initial exposure is 10 years or more, may reasonably be monitored with chest films and pulmonary function studies every 3 to 5 years to identify the onset of asbestos-related disease.

Persons with a history of exposure to asbestos are also at risk for asbestos-related malignancies. Periodic health surveillance for lung cancer or mesothelioma is not recommended. Screening for lung cancer using periodic (annual) chest films, low-dose computed tomography, or sputum cytology has not been shown to be effective in preventing mortality or improving quality of life in populations of smokers without known adverse occupational exposures (152, 153). New technologies (e.g., low-dose spiral CT scanning) are being evaluated for use in high-risk groups (153). The risk of extrathoracic malignancies may also be increased in asbestos-exposed workers. Studies suggest that there may be an elevation in the risk of colon cancer (149, 150), although this remains controversial (154). Because colon cancer is often treatable and screening for colorectal cancer is recommended by the American Cancer Society for persons more than 50 years of age (155), it is reasonable on the basis of current evidence to screen for this condition. The risk of cancer of the larynx (156) and possibly gastrointestinal cancers other than colon, including pancreas, stomach, and esophagus (154), may also be increased with asbestos exposure, but the presence and magnitude of an association with asbestos remain controversial for extrathoracic cancers (154). Routine screening for these cancers is in any case not practical at present.

No prophylactic medication or treatment is currently available to prevent the development or progression of asbestosis or other asbestos-related diseases, once exposure has occurred.

Actions Required after Diagnosis

The diagnosis of asbestosis, in particular, imposes a duty to inform the patient that he or she has a disease that is work-related, to report the disease, and to inform the patient that he or she may have legal or adjudication options for compensation. The role of the physician in this compensation process includes performing an objective evaluation of impairment consistent with the rules of the specific compensation system. Guidelines developed by the American Thoracic Society (3) may be of use and are incorporated into the *AMA Guides to the Evaluation of Permanent Impairment* (157). As in the management of any lung disorder, the physician should also manage the clinical manifestations of the disease and counsel the patient to protect remaining lung function.

The patient with evidence of asbestosis should be considered to be at risk of progressive lung disease, whatever the level of impairment on first encounter. It seems logical that removal from further exposure to asbestos or other significant occupational and environmental exposures may avoid more rapid progression of lung disease, although specific evidence for this is lacking. However, if such exposures are minimal and are well within occupational guidelines, care must be taken not to deprive the patient of a livelihood for no clinical benefit.

Immunization against pneumococcal pneumonia and annual influenza vaccine should be administered unless contraindicated for other reasons. Effective management of concurrent chronic obstructive pulmonary disease or asthma, if present, may reduce morbidity from mixed disease.

Severe asbestosis is rare in the United States and other countries with generally effective occupational health regulation. Cor pulmonale, secondary polycythemia, and respiratory insufficiency and failure are all treated in the conventional manner in patients with asbestosis.

In the spring of 2000, the Association of Occupational and Environmental Clinics adopted a resolution recommending necessary standards for screening programs (158). This action was taken in response to the proliferation of screening programs undertaken to identify cases for possible legal actions in which counseling and education may be lacking (159), but the recommendations also apply to those conducted for patient care and protection. Their recommendations were consistent with those given above and also emphasized timely physician disclosure of results to the patient, appropriate medical follow-up, and patient education. The National Institute of Occupational Safety and Health has outlined elements of an adequate screening program, with special reference to screening for asbestos-related disorders in currently employed mineworkers, in a white paper produced in 2002 that has received little attention (160). The National Institute for Occupational Safety and Health recommended that such programs should be under the direction of a "qualified physician or other qualified health care provider" knowledgeable in the field and competent to administer it, and documented with written reports to workers and employers (the latter provision that would not necessarily be applicable to workers who had separated from the employer). However, the National Institute for Occupational Safety and Health did not address the issue of counseling in that document or clinical interventions to reduce future risk.

CONCLUSIONS

The diagnosis of nonmalignant asbestos-related disease rests, as it did in 1986, on the essential criteria described: a compatible structural lesion, evidence of exposure, and exclusion of other plausible conditions, with an additional requirement for impairment assessment if the other three criteria suggest asbestos-related disease (2). Each criterion may be satisfied by one of a number of findings or tests. The 2004 criteria are open to future testing modalities if and when they are validated. For example, HRCT has greatly increased the sensitivity of detection and has become a standard method of imaging. Evidence for exposure still rests on the occupational history, the demonstration of asbestos fibers or bodies, or pleural plaques. Impairment evaluation is largely unchanged from 1986 and remains an essential part of the clinical assessment. Potentially confounding conditions, such as idiopathic pulmonary fibrosis, are better understood and many, such as tuberculosis, are less common than in the past so that the clinical picture is less often confusing.

These criteria and the guidelines that support them are compatible with the Helsinki criteria, developed by an expert group in 1997, which represents substantial consensus worldwide (147). The guidelines supporting these criteria will undoubtedly change again in future, but the present guidelines should provide a reliable basis for clinical diagnosis for some years to come.

This statement was developed by an ad hoc subcommittee of the Scientific Assembly on Environmental and Occupational Health of the American Thoracic Society. Members of the committee are as follows:

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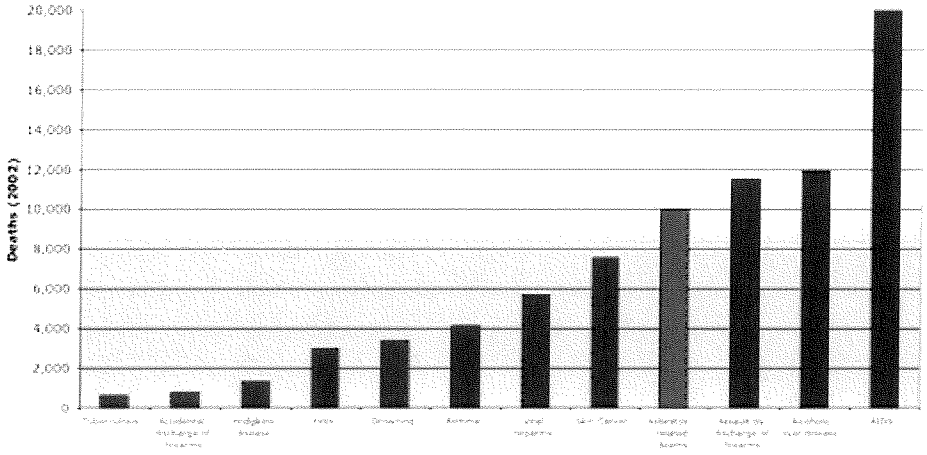
The Asbestos Epidemic in America

The Asbestos Industry lobby attack in Washington and now Lansing over asbestos litigation has overshadowed a quiet and directly related crisis in public health: an epidemic of asbestos-caused diseases in the United States that claims the life of one out of every 125 American men who die over the age of 50.

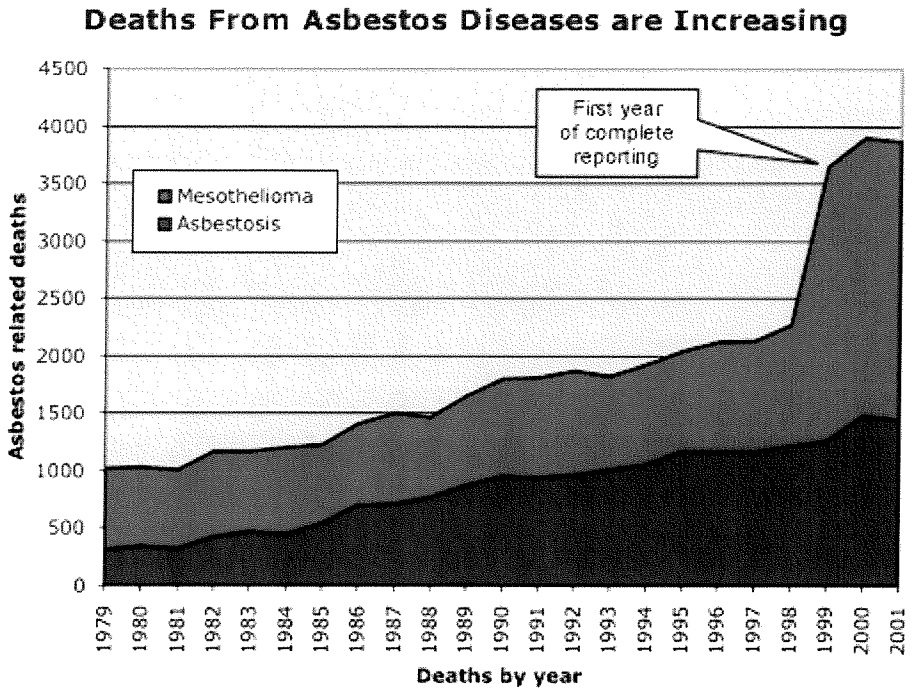
Ten thousand Americans die each year -- a rate approaching 30 deaths per day -- from diseases caused by asbestos, according to a detailed analysis of government mortality records and epidemiological studies by the EWG Action Fund. Asbestos kills thousands more people than skin cancer each year, and nearly the number that are slain in assaults with firearms. The suite of diseases linked to asbestos exposure overwhelmingly affect older men.

Asbestos-related deaths are at an epidemic scale in the United States



(asbestos in red) source EWG

Even more disturbing, deaths from asbestos in the United States appear to be increasing. Mesothelioma and asbestosis mortality rose steadily from 1979 through 1998. Asbestosis mortality, however, rose at more than three times the rate of mesothelioma, at 7.8 percent per year, compared to 2.3 percent annually for mesothelioma over the 24-year period 1979-2001.



Source: EWG Action Fund. Compiled from Centers for Disease Control and Prevention (CDC), National Center for Health Statistics (NCHS), multiple cause of death file 1979-2001. Does not include asbestos-caused mortality from lung or gastrointestinal cancer.

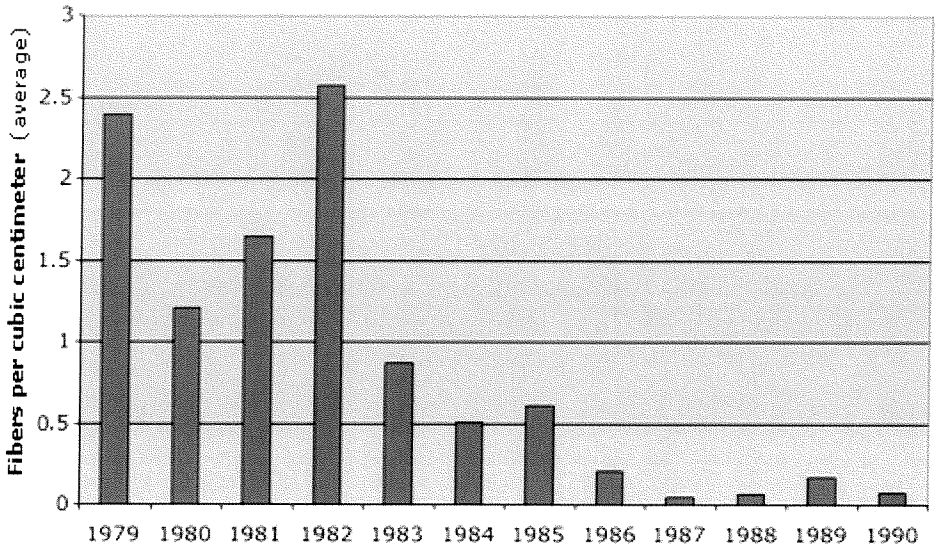
As in the United Kingdom (Treasure 2004) and Australia (Leigh 2003), there are many reasons to believe that the peak of the U.S. asbestos disease epidemic may not be reached for a decade or more.

Asbestos use and exposure crested in the United States in the mid 1970s when a number of factors converged: more than 3,000 consumer and industrial products on the market at that time contained asbestos; asbestos product factories were polluting nearby neighborhoods; asbestos workers were heavily exposed on the job and were bringing home substantial amounts of asbestos dust to their wives and children; and asbestos was commonly used in public buildings and workplaces for soundproofing, fireproofing, and insulation. Meaningful workplace safeguards were not in place until at least 1980, and for many industries, such as construction, levels in excess of the pre-1980 standard persist even today (NIOSH 2002).

Asbestos diseases have a 20 to 50 year latency period, meaning that a substantial portion of individuals exposed in the 1960s and 1970s are just now showing up as disease or mortality statistics. Better tracking accounts for the dramatic increase in mesothelioma mortality reported in 1999, but lung cancer deaths from asbestos are not reported at all, and asbestosis is still dramatically underreported even in worker populations where asbestos exposure is well established (Markowitz

1997). And asbestos has not been banned. It remains used in some brake shoes and other products, directly exposing auto mechanics and others who work with the materials, and indirectly exposing consumers and workers' families. In addition, millions of people are exposed at home or in their workplace by the monumental quantities of asbestos that remain in the built environment -- the attic insulation in 30 million American homes, for instance -- following decades of heavy use.

Asbestos exposures remained high through the early 1980s

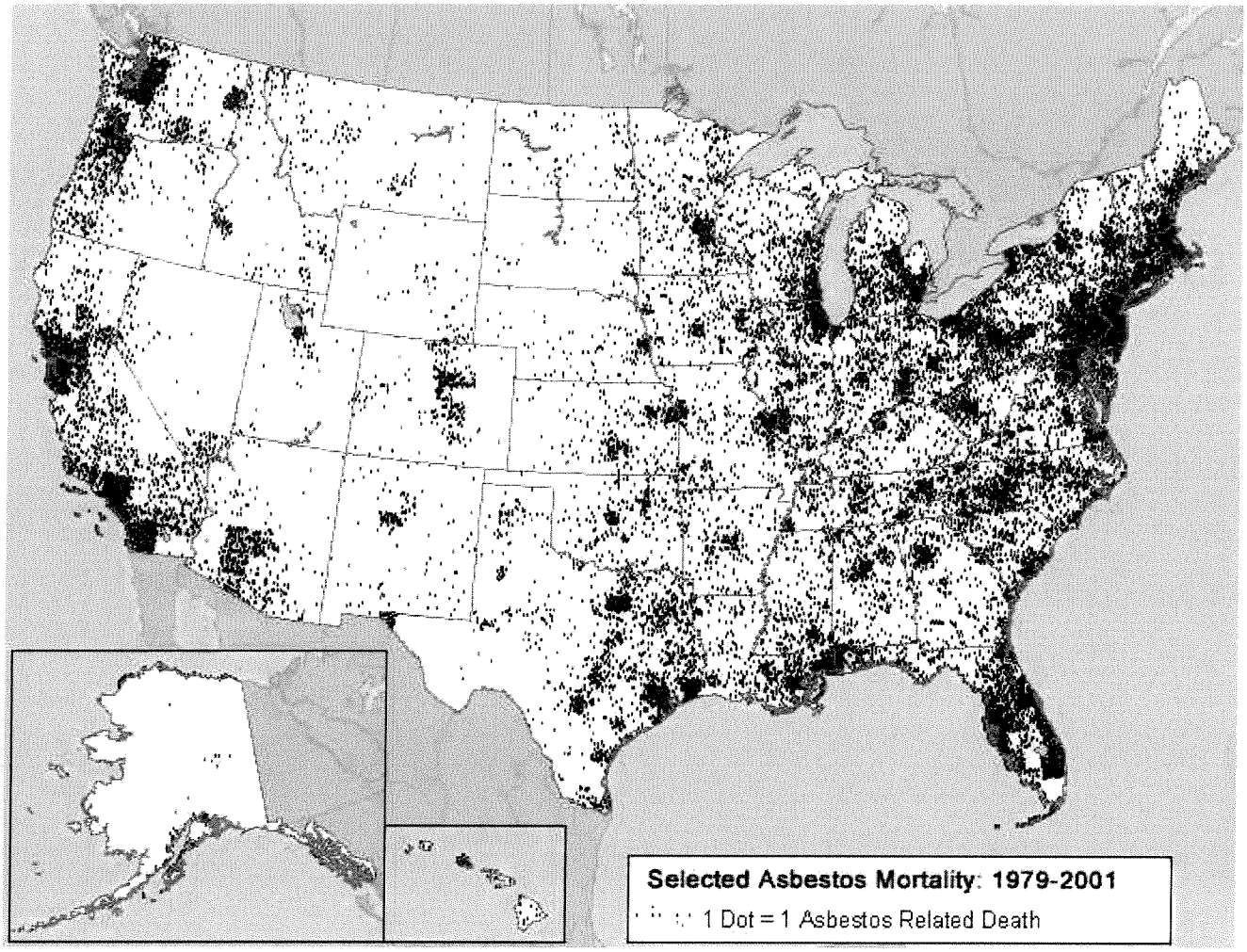


Source: EWG Action Fund, compiled from Occupational Safety and Health Administration health inspection data (1979 - 1998). Data includes 19,000 samples from 670 industries.

We would suggest the Michigan Senate consider banning asbestos product sales rather than asbestos victim lawsuits.

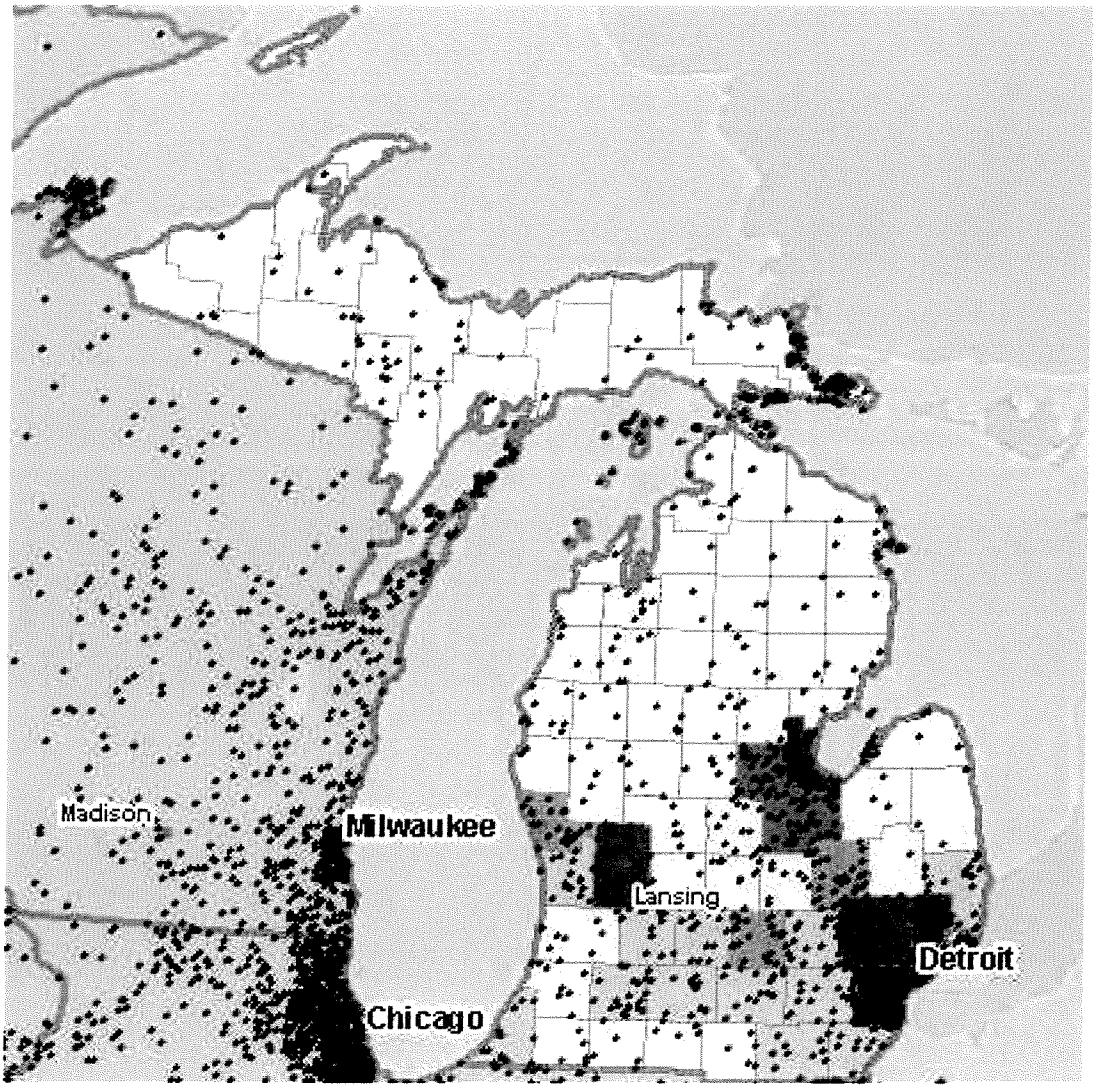
United States Government Data on Asbestos Mortality

43073 people killed by asbestos from 1979 through 2001



Asbestos deaths from Mesothelioma or asbestosis as reported to the federal government via death certificate records from 1979 through 2001.

Asbestos Mortality in Michigan



There have been at least 1,140 deaths caused by asbestos exposure in Michigan between 1979 and 2001.

Asbestos Corporate Knowledge Timeline (Condensed)

Asbestos Hazards Recognized

1890s Asbestos, which previously had few industrial uses, becomes a raw material for large manufacturing industries, exposing large numbers of workers to asbestos dust for the first time. Asbestos-caused disease often develops decades after a person was first exposed. As a result, it was not until the early 1900s that large numbers of workers developed symptoms.
David Kotelchuck, "Asbestos: 'The Funeral Dress of Kings' - and Others" in Dying for Work: Workers' Safety and Health in Twentieth-Century America, ed. by David Rosner and Gerald Markowitz, Indiana University Press, Bloomington, IN 1987, p193

1918 A Prudential Insurance Company official notes that life insurance companies will not cover asbestos workers, because of the "health-injurious conditions of the industry."
Barry I. Castleman, Asbestos: Medical and Legal Aspects, 4th edition, Aspen Law and Business, Englewood Cliffs, NJ 1996, p.5-6

1930 Major asbestos company Johns-Manville produces report, for internal company use only, about medical reports of asbestos worker fatalities.
Barry I. Castleman, Asbestos: Medical and Legal Aspects, 4th edition, Aspen Law and Business, Englewood Cliffs, NJ 1996, p.663

1932 Letter from U.S. Bureau of Mines to asbestos manufacturer Eagle-Picher states: "It is now known that asbestos dust is one of the most dangerous dusts to which man is exposed."
Paul Brodeur, Outrageous Misconduct: The Asbestos Industry on Trial, Pantheon Books, New York NY, 1985, p.327

1933 Metropolitan Life Insurance Co. doctors find that 29 percent of workers in a Johns-Manville plant have asbestosis.
Barry I. Castleman, Asbestos: Medical and Legal Aspects, 4th edition, Aspen Law and Business, Englewood Cliffs, NJ 1996, p.26

Johns-Manville officials settle lawsuits by 11 employees with asbestosis on the condition that the employees' lawyer agree to never again "directly or indirectly participate in the bringing of new actions against the Corporation."
Paul Brodeur, Outrageous Misconduct: The Asbestos Industry on Trial, Pantheon Books, New York NY, 1985, p.114

1934 Officials of two large asbestos companies, Johns-Manville and Raybestos-Manhattan, edit an article about the diseases of asbestos workers written by a Metropolitan Life Insurance Company doctor. The changes minimize the danger of asbestos dust.
Paul Brodeur, Outrageous Misconduct: The Asbestos Industry on Trial, Pantheon Books, New York NY, 1985, p.114-15

Denial, Deception and Document Suppression

1935 Officials of Johns-Manville and Raybestos-Manhattan instruct the editor of Asbestos magazine to publish nothing about asbestosis.
Paul Brodeur, Outrageous Misconduct: The Asbestos Industry on Trial, Pantheon Books, New York NY, 1985, p.116

1936 A group of asbestos companies agrees to sponsor research on the health effects of asbestos dust, but require that the companies maintain complete control over the disclosure of the results.
Barry I. Castleman, Asbestos: Medical and Legal Aspects, 4th edition, Aspen Law and Business, Englewood Cliffs, NJ 1996, p.587

1942 Internal Owens-Corning corporate memo refer to "medical literature on asbestosis . . . . scores of publications in which the lung and skin hazards of asbestos are discussed."
Barry I. Castleman, Asbestos: Medical and Legal Aspects, 4th edition, Aspen Law and Business, Englewood Cliffs, NJ 1996, p.195

1942 or 1943 The president of Johns-Manville says that the managers of another asbestos company were "a bunch of fools for notifying employees who had asbestosis." When one of the managers asks, "do you mean to tell me you would let them work until they dropped dead?" The response is reported to have been, "Yes. We save a lot of money that way."
Testimony of Charles H. Roemer, Deposition taken April 25, 1984, Johns-Manville Corp., et al v. the United States of America, U.S. Claims Court Civ. No. 465-83C, cited in Barry I. Castleman, Asbestos: Medical and Legal Aspects, 4th edition, Aspen Law and Business, Englewood Cliffs, NJ 1996, p.581

1944 Metropolitan Life Insurance Company report finds 42 cases of asbestosis among 195 asbestos miners.
Barry I. Castleman, Asbestos: Medical and Legal Aspects, 4th edition, Aspen Law and Business, Englewood Cliffs, NJ 1996, p.654

1951 Asbestos companies remove all references to cancer before allowing publication of research they sponsor.
Barry I. Castleman, Asbestos: Medical and Legal Aspects, 4th edition, Aspen Law and Business, Englewood Cliffs, NJ 1996, p.71

1952 Dr. Kenneth Smith, Johns-Manville medical director, recommends (unsuccessfully) that warning labels be attached to products containing asbestos. Later Smith testifies: "It was a business decision as far as I could

understand . . . the corporation is in business to provide jobs for people and make money for stockholders and they had to take into consideration the effects of everything they did and if the application of a caution label identifying a product as hazardous would cut into sales, there would be serious financial implications."
Barry I. Castleman, Asbestos: Medical and Legal Aspects, 4th edition, Aspen Law and Business, Englewood Cliffs, NJ 1996, p.666

1953 National Gypsum's safety director writes to the Indiana Division of Industrial Hygiene, recommending that acoustic plaster mixers wear respirators "because of the asbestos used in the product." Another company official notes that the letter is "full of dynamite," urges that it be retrieved before reaching its destination. A memo in the files notes that the company "succeeded in stopping" the letter, which "will be modified."
Barry I. Castleman, Asbestos: Medical and Legal Aspects, 4th edition, Aspen Law and Business, Englewood Cliffs, NJ 1996, p.669-70

1964 Dr. Irving Selikoff publishes a study of asbestos workers in the Journal of the American Medical Association, proving that people who work with asbestos-containing materials have an abnormal incidence of asbestosis, lung cancer, and mesothelioma.
Barry I. Castleman, Asbestos: Medical and Legal Aspects, 4th edition, Aspen Law and Business, Englewood Cliffs, NJ 1996, p.126

1966 Raybestos-Manhattan official writes: "We feel that the recent unfavorable publicity over the use of asbestos fibers in many different kinds of industries has been a gross exaggeration of the problems. There is no data available to either prove or disprove the dangers of working closely with asbestos."
Barry I. Castleman, Asbestos: Medical and Legal Aspects, 4th edition, Aspen Law and Business, Englewood Cliffs, NJ 1996, p.590

"My answer to the problem is: if you have enjoyed a good life while working with asbestos products why not die from it. There's got to be some cause."
Sept 12, 1966 Document E.A. Martin, Honeywell-Bendix

1971 First OSHA asbestos-exposure standard issued.
Federal Register, vol.36, p.10466 et. seq.; May 29, 1971

1973 The U.S. Environmental Protection Agency (EPA) bans spray-on asbestos insulation as an air pollution hazard.
Federal Register, vol.38, p.8820 et. seq.; April 6, 1973

1977 Lawyers for injured workers obtain the Sumner Simpson papers, which show that the companies had suppressed information about the danger of asbestos for at least 40 years.
Paul Brodeur, Outrageous Misconduct: The Asbestos Industry on Trial, Pantheon Books, New York NY, 1985, p.111
*The first bill to limit the product liability of asbestos companies is introduced in Congress.
Paul Brodeur, Outrageous Misconduct: The Asbestos Industry on Trial, Pantheon Books, New York NY, 1985, p.194

1978 Judge rules there had been "a conscious effort by the [asbestos] industry in the 1930s to downplay or arguably suppress, the dissemination of information to employees and the public for fear of the promotion of lawsuits."
Amended order, Barnett v. Owens-Corning Fiberglas Corp et al, State of South Carolina, County of Greenville, Court of Common Pleas, Aug. 23, 1978, cited in Barry I. Castleman, Asbestos: Medical and Legal Aspects, 4th edition, Aspen Law and Business, Englewood Cliffs, NJ 1996, p.585

1979 U.S. EPA announces intention to issue rule that bans all uses of asbestos. (10 years to complete)
Federal Register, vol 44, p.60061

1982 Johns-Manville files for bankruptcy protection.
Paul Brodeur, Outrageous Misconduct: The Asbestos Industry on Trial, Pantheon Books, New York NY, 1985, p.249

1986 OSHA tightens asbestos-exposure standard.
Federal Register vol. 51, p.22733, June 20, 1986
U.S. EPA publishes text of proposed rule to ban all uses of asbestos.
Environmental Defense Newsletter, XVII:2 May 1986

1989 The U.S. EPA bans asbestos in most of its major uses, but . . .
Federal Register, vol.59, p.41027, August 10, 1994

1991 Asbestos companies win federal lawsuit, court revokes EPA's 1989 asbestos ban.
Federal Register, vol.59, p.41027, August 10, 1994

1994 OSHA tightens asbestos-exposure standard.
Federal Register, vol. 59, p.40964 et. seq., August 10, 1994

1999 Florida Supreme Court rules that Owens Corning willfully withheld information about the danger of working with the company's asbestos products: "It would be difficult to envision a more egregious set of circumstances . . . . a blatant disregard for human safety involving large numbers of people put at life-threatening risk."
Opinion No. 92,963, August 26, 1999

For the Asbestos Industry, Bankruptcy Means "Business as Usual"

Just as the ongoing epidemic of asbestos-caused mortality and injury has been overshadowed by the controversy over litigation, the origins of asbestos lawsuits have been buried beneath claims of the Asbestos Industry and Chamber of Commerce Lobbyists that litigation has "bankrupted" dozens of large U.S. companies, and that the Michigan Legislature and the Michigan Supreme Court must put an end to or severely limit asbestos litigation in order to protect more companies from going bankrupt in the future.

But neither asbestos lawsuits, nor contemporary business group proposals to end it, should have been necessary at all. The controversy could have been avoided if the very same companies now pressing for "asbestos litigation reform" had acted responsibly and compassionately decades ago, when their highly detailed, proprietary knowledge showed that asbestos posed mortal risks to millions of their workers, and to tens of millions of Americans who came in contact with the deadly substance in their homes, schools and workplaces.

Instead of fair and respectful consideration for their workers and others, asbestos and insurance companies offered only cold, unrelenting resistance. The companies aggressively fought requests for financial or medical aid and support; they callously, and notoriously, hid unambiguous scientific evidence of asbestos exposure, injury and death. Indeed, no meaningful proposals for help of any kind were forthcoming from asbestos industries and their insurers until a handful of people, out of hundreds of thousands whose lives had been destroyed by asbestos illnesses and death, went to court seeking justice because they had no other choice -- and began to win.

Proponents of Legislative action to block asbestos lawsuits now argue that this litigation must be stopped because it is "bankrupting" asbestos companies, their insurers, and by some accounts, the entire U.S. economy. Political speeches, and many media accounts, routinely refer to companies that are "bankrupt" as a result of asbestos litigation. As this section explains, "bankruptcy" connotes a degree of business distress that is rarely experienced by companies that have been sued by people with asbestos-related health problems.

According to Halliburton, it's "business as usual" as a result of their asbestos bankruptcy:

> Q. In Europe and many other countries, when a company is "bankrupt," it means that it is going out of business. What is different here?
>
> A. European bankruptcy laws, as in many countries, are very different from the laws in the U.S. Chapter 11 has been created so that a filing company can restructure its debt (or in our case resolve its asbestos and silica liability) and remain in business. It is not a liquidation; it is a reorganization.
>
> Halliburton and all of its subsidiaries, including DII Industries and KBR, will continue in business and will continue to provide all the excellent services our customers expect from us. The Chapter 11 petitions have been filed for the sole purpose of facilitating a settlement of Halliburton's personal injury asbestos and silica litigation claims. In other words, outside of the asbestos and silica settlement, it will be business as usual.

When most people hear that a company is going bankrupt, they think liquidation of assets, massive layoffs, and shutting down the business. With asbestos bankruptcies this is the very rare exception. Most "bankrupt" asbestos companies, especially the larger corporations typically offered as examples of asbestos-induced economic havoc, remain very competitive within their industries during bankruptcy, and often flourish afterwards.

This is because an asbestos bankruptcy is a reorganization authorized under Chapter 11 of the bankruptcy code, not a liquidation that occurs under Chapter 7. It is a way to stop ongoing and future litigation, consolidate liability, and protect the company and all of its subsidiaries from future liability. While not painless, it is a relatively smooth and equitable way for a company to assist the families of workers and others injured or killed by asbestos.

The asbestos industry and its supporters use the popular image of bankruptcy to argue that aiding people hurt by asbestos is costing huge numbers of jobs, ravaging the pension plans of innocent workers, and bankrupting the economy.

A look at websites of "bankrupt" companies reveals a very different assessment of the economic and financial impact of asbestos bankruptcies.

Halliburton calls its asbestos bankruptcy "good news" and a "definite win for people who care about Halliburton." (see www.halliburton.com/ir/asbestos_faqs.jsp

The company certainly does not feel that its $4.5 billion settlement with asbestos victims represents any threat to the ongoing profitability of the company.

To quote again from their website:

> In a successful implementation of an asbestos settlement under Chapter 11, most aspects of the company's business do not have to change. Under the proposed Plan of Reorganziation (sic):
>
> • The company and its subsidiaries do not go out of business.
>
> • Nothing necessarily changes at any business units, whether they are in Chapter 11 proceedings or not, from an operational standpoint.
>
> • No facilities need to close and no jobs need to be eliminated as a result of a Chapter 11 filing.
>
> • No pension or benefits programs are be (sic) reduced or eliminated.
>
> • No employees have their salaries cut, or promotion opportunities restricted.
>
> • No vendors are delayed in payment from normal terms.
>
> • No creditors are delayed in payment from normal schedule.
>
> • No business units outside the U.S. are affected in any way.
>
> • The company does not have to renegotiate contracts as a result of the Chapter 11 filing.
>
> From www.halliburton.com/ir/chapter11_primer.jsp

In 1994, Congress amended the bankruptcy code to provide special protection for companies with asbestos liability. What distinguishes these amendments from traditional Chapter 11 bankruptcy is that they allow companies to seek bankruptcy protection from *future* liability, if they can show that future liability exceeds the assets of the company (Bankruptcy Reform Act of 1994). As a result of these changes, bankruptcy emerged as the preferred option for companies seeking to limit asbestos liabilities. Forty-eight firms filed for bankruptcy due to asbestos claims

between 1982 and 1999, and an additional thirty firms filed between 2000 and 2002 (White, 2002, at 1320).

The 1994 amendments are known as the "Manville Amendments," because they were modeled after the core components of the Manville Trust, a legal entity established to settle asbestos claims against a major asbestos company, Johns-Manville. In addition to allowing asbestos companies to settle all future asbestos liability claims by filing Chapter 11 and establishing a special asbestos bankruptcy trust, known as a 524(g) trust, the law also grants courts the power to issue injunctions that prevent all asbestos litigation against the company and its subsidiaries from moving forward, a major benefit to companies with substantial liabilities (Bankruptcy Reform Act of 1994; Macchiarola, 1996, at 617). The amendments also applied retroactively to litigation that was ongoing at the time of passage (Crames, et al., 2002; White, 2002, at 1322).

In practice, Chapter 11 asbestos bankruptcies rarely result in lost jobs or diminished pensions beyond what would be attributable to the normal business cycle. Instead, the Chapter 11 bankruptcy allows a company to receive an "automatic stay," which stops all payments to creditors (including payments owed through settlements), stops all pending lawsuits, and lets the company reorganize and then prioritize payments.

Under the Chapter 11, section 524(g), an asbestos company can stop all of its pending asbestos lawsuits and set up a fund to settle all present and future asbestos claims. This automatic stay provision also extends to parent and subsidiary companies and protects them from future asbestos lawsuits. Because Chapter 11 requires the company to adopt a court-approved reorganization plan, payments on asbestos claims may be delayed as long as five to six years while the plan is developed, approved, and implemented (White, 2002, at 1320).

While any form of bankruptcy is serious, it is clear that asbestos filings under Chapter 11 have not wreaked havoc on the economy. Between February 2000 and October 2001, the seven largest companies facing asbestos liability filed for bankruptcy protection under Chapter 11. These companies include Babcock & Wilcox, Owens Corning, Armstrong, W.R. Grace & Co., U.S. Gypsum Co., Federal-Mogul and Building Materials Corporation of America.

An analysis of 10K filings for these companies for the years 1998 through 2002 concluded that:

> "The Chapter 11 companies have been able to continue operations successfully. Indeed, with few exceptions, they have prospered, increasing their sales. They have been able to maintain their assets and employment, meet their obligations to business creditors and employees, and make capital investments that will allow them to continue to prosper." (Benston, 2003, at 5).

A review of the companies' public statements confirms this conclusion. Babcock & Wilcox filed for bankruptcy in 2002. The company explains its decision to file for Chapter 11 protection from asbestos liability as follows:

> When a company files for Chapter 11, it is permitted to continue operating while developing a plan to emerge as a stronger, healthier company. Most people, when they hear "bankruptcy," think "liquidation" (that is, when a company sells off all its assets and inventory and goes out of business). That is a different kind of bankruptcy, called a Chapter 7. Chapter 11 does not mean liquidation.
>
> B&W's core business continues to be strong. B&W filed for protection under Chapter 11 because it offers the only viable legal process for determining and comprehensively resolving its asbestos claims.
>
> B&W's core operating business continues to be a solvent and strong business with a backlog totaling over $1 billion. [t]here should be little impact on day-to-day operations. It's business as usual. Project work will continue. [w]e expect there will be no effect on salaries, benefits or promotion opportunities.
>
> See www.babcockwilcox.com/pgg/pr/chapter11.html.

Another asbestos manufacturer, Owens-Corning, filed for bankruptcy protection on October 12, 2000 to settle its approximately $2 billion in asbestos liability. The company had previously paid out or had commitments to pay out $5 billion. (Asbestos Litigation Reporter, 2002).

Senate Majority leader Frist named Owens-Corning on the floor of the Senate as a company that had been driven to bankruptcy by excessive asbestos litigation and then went on to say that:

> "Asbestos-related bankruptcies spell doom for these workers' jobs; thus, their families, and, of course, incomes and retirement savings."

Owens-Corning has a dramatically different take on its Chapter 11 asbestos proceeding:

> On Thursday, October 5, 2000, Owens Corning voluntarily filed a petition for reorganization under Chapter 11 bankruptcy protection in the United States Bankruptcy Court in Wilmington, Delaware.
>
> The filing will enable the company to refocus on operating its business and serving its customers, while it develops a plan of reorganization that will resolve its asbestos and other liabilities and provide a suitable capital structure for long-term growth.
>
> To enhance its liquidity, Owens Corning has obtained a $500 million debtor-in-possession financing commitment from Bank of America. Upon court approval, these funds will be available to the company to help meet its future needs and fulfill obligations associated with operating its business, including payment under normal terms to suppliers and vendors for all goods and services that are provided after today's filing. Employees will continue to be paid in the normal manner and their health benefits, as well as those of retirees, will not be disrupted. The company's pension plan for retirees and vested employees is fully funded and protected by federal law.
>
> It is important for our customers and business partners to know that all Owens Corning operations are open and we are continuing to focus on serving our customers. Customer service and daily operations are our top priorities.
>
> See www.owenscorning.com/financialreorganization/.

Owens-Corning's Chairman and CEO, Glen Hiner, assessed the impact of the company's asbestos bankruptcy filing this way:

> "[w]ith the Chapter 11 process we can finally put this difficult issue behind us in a fair and responsible manner and move forward with our resources and energies focused on competing successfully in the global marketplace." Cy Goldberg & Dareen J. Check, Bullseye Gets Bigger on Peripheral Defendants: The Effect of Bankruptcies on Asbestos Litigation, The Legal Intelligencer (Apr. 25, 2001).

Clearly these companies have not "gone bankrupt" in the sense commonly imagined by the public and invoked by politicians who are pressing for "asbestos litigation reform." Instead, these asbestos companies have taken advantage of a special provision of Chapter 11 called a 524(g) trust, which was specifically inserted into the tax code by the Congress in 1994 to help asbestos manufacturers shield current assets from present and future asbestos liability (Green Testimony, 2003).

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3. Frank J. Macchiarola, The Manville Personal Injury Settlement Trust: Lessons for the Future, 17 Cardozo L. Rev. 583 (1996).

4. Michael J. Crames, Benjamin Mintz & Dean M. Trafelet, Section 524(g) & the Futures Representative, ALI-ABA Course of Study Materials, Asbestos Litigation in the 21st Century (Sept. 2002).

5. Asbestos Liability Claims Cause Owens-Corning to File for Chapter 11, 22 Asbestos Litigation Reporter 3 (Oct. 12, 2002).

6. Testimony of Prof. Eric D. Green, U.S. Senate, Judiciary Committee Hearing on S. 1125 (June 4, 2003).

7. EWG.org

Mogul Jumping

Federal Mogul: Wheeling & Dealing in Asbestos Liability

"We believe today we have a sound operational business plan, a clear and exciting strategy, and the elements of a restructuring plan that will allow us to emerge from Chapter 11 as a financially strong company with a solid future."

— Federal-Mogul CEO speaking at Annual Shareholders Meeting, 5/15/02. See: http://www.federal-mogul.com/cda/content/front/0,2194,2336__4083,00.html

The Federal-Mogul company presents a tortuous case study of a corporation that knowingly became enmeshed in asbestos liability as a business acquisition strategy; entered into the development of a traditional asbestos reorganization plan to deal with asbestos claims; aggressively sought a bail-out from Congress to reduce those liabilities in lieu of a reorganization plan; and finally, and forcefully, rejected the Frist-Hatch bill (S. 2290) as unfairly costly to the company.

Federal-Mogul is a multinational auto-parts conglomerate serving auto manufacturers and the aftermarket. Its world headquarters are in Southfield, Michigan. Federal-Mogul employs over 45,000 people worldwide, and its better-known product lines include Champion spark plugs, Wagner lighting systems and brake pads, and ANCO wiper blades. Federal-Mogul reports 2004 revenues of over $5.5 Billion.
(See company website: http://www.federal-mogul.com/cda/content/front/0,2194,2548_2947_16194,00.html)

Federal-Mogul has spent the past few years attempting to clear its acquired asbestos liabilities, first through lobbying Congress for a bailout, and when that failed, through filing for Chapter 11 bankruptcy protection. Now they have brought their attack to Lansing.

History of Federal-Mogul's Asbestos Involvement

Federal-Mogul had very limited involvement with asbestos prior to its acquisition of several companies with significant asbestos liabilities in the late 1990s. Those acquisitions, which catapulted the company onto the Fortune 500 list for the first time in 1998 (ranked #349), gave the company a much stronger foothold within its industry. However, along with that added market strength and rapid growth came huge asbestos liabilities acquired during the purchase of companies which had made asbestos products and been sued by customers and others.

The company explains the history of its acquired asbestos liabilities on its website:

"There are essentially seven different streams of asbestos liability affecting Federal-Mogul, each arising from a Federal-Mogul acquisition of a discrete company with its own unique role in the manufacture, distribution and sale of asbestos-containing materials."

(See: http://www.federal-mogul.com/cda/content/front/0,2194,2336_2903_4292,00.html)

The largest source of asbestos liability resulted from Federal-Mogul's acquisition of British automotive supplier T&N Plc, which formerly manufactured auto parts containing asbestos. However, Federal-Mogul also acquired several other companies within the same time frame that had significant asbestos liabilities. Given their scale and number, it is difficult to believe these acquisitions were anything other than strategic business decisions.

Federal-Mogul acknowledges today on its website that, prior to those acquisitions, the company was fully aware of the asbestos liabilities involved, yet proceeded with the acquisitions with the confidence that it could handle the consequences.

The company website explains:

23. What caused the asbestos liability?

Federal-Mogul inherited most of its asbestos liability through its acquisition of T&N plc, a U.K. company, and its subsidiaries in March 1998. T&N, formerly known as Turner & Newall, was among other things, an English building materials manufacturer that used and sold asbestos products. When Federal-Mogul acquired T&N, it no longer manufactured these materials but was a recognized manufacturer of automotive products headquartered in Manchester, England.

24. Did you know about the asbestos liability at the time of the acquisition?

Yes. The company set up a reserve in March 1998 for approximately $2.1 billion (including insurance) to handle the estimated asbestos liability post acquisition. This anticipated asbestos liability was based on work done to put in place an asbestos insurance policy in late 1996.
(See: http://www.federal-mogul.com/cda/content/front/0,2194,2336__4270,00.html)

After the flurry of acquisitions was complete, Federal-Mogul began lobbying Congress "vigorously" for a legislative solution to its asbestos "issues." Frank Macher, former CEO and Chairman of the Board of Federal-Mogul, explained the strategy in a Federal-Mogul Annual Shareholders Meeting:

"Along with this new litigation approach, we took a leadership role in seeking a legislative solution, co-chairing a group of 30 other Fortune 500 companies with asbestos issues creating an active alliance. Working together, we sought sponsorship and support of legislation that provided for medical criteria, venue criteria, and limited the consolidation of cases.

We also intensified the senior management team's involvement with the asbestos issue. We established an Asbestos Strategy Review Board, comprised of many of our

executives and chaired by me. The handling of our asbestos issues was a high priority and we focused many, many hours on both our litigation strategy and our legislative efforts.

Jim Zamoyski and I traveled regularly to Washington, D.C. seeking legislative support from U.S. Senators and members of Congress. We launched an aggressive letter-writing campaign that energized our U.S. employees, suppliers and customers for the legislative cause. I thank all shareholders who participated in the letter writing campaign.

Just as it seemed that we had the momentum and support going our way, changes in the U.S. Senate leadership dramatically halted our progress. In fact, NAM, the National Association of Manufacturers, took over the leadership of this coalition last summer and they are still working to have a bill introduced."
(Frank Macher 5/15/02. See: http://www.federal-mogul.com/cda/content/front/0,2194,2336__4083,00.html)

When their lobbying efforts failed, Federal-Mogul filed for Chapter 11 protection on October 1, 2001 to shield their business from further litigation. Mr. Macher explained:

"The decision to file was extremely difficult. But we could not allow the enterprise value of Federal-Mogul to be drained any further. On October 1st, we acted to separate our asbestos liabilities from the company's true operating potential by voluntarily filing for financial restructuring in Bankruptcy Court in the United States and Administration in the United Kingdom."
(Frank Macher 5/15/02. See: http://www.federal-mogul.com/cda/content/front/0,2194,2336__4083,00.html)

Some stock analysts and financial reporters stated that Federal Mogul was driven to bankruptcy not primarily by its asbestos liabilities, but by its overzealous acquisition strategy years prior under previous leadership. Federal-Mogul announced 13 acquisitions in 1998 alone.

"Some analysts had seen Federal-Mogul as a candidate for bankruptcy court as the company struggled under a mountain of debt acquired during a $6-billion buying spree in 1998."
(Detroit Free Press, "Credit Gives Big Supplier A Life Federal-Mogul Says Added $550 Million Assures Its Survival," Jan. 4, 2001)

"Asbestos-related claims are only one of the many problems Federal-Mogul faces, however. Even without the asbestos situations, the company's core business units have been faltering.

After a long string of acquisitions in the late 1990's, F-M top management was widely criticized for failing to integrate the businesses and capitalize on the potential synergies. In the ultra-competitive automotive OEM and aftermarket business arenas, competitors quickly learned to capitalize on Federal-Mogul's weaknesses."

(eBearing News, "Federal-Mogul Files Chapter 11 Bankruptcy," Oct. 2, 2001. See: http://www.eBearing.com/news2001/news341.htm)

"The company blames a soft market for spare parts, but rivals do not seem affected and US car sales are booming. It is more likely that Federal-Mogul is having trouble integrating its hasty purchases. The merger machine is starting to look like a takeover target."
(Financial Times, "Federal-Mogul," September 16, 1999)

Federal-Mogul's creditors reportedly have felt the company was so sure it could get Congress to solve its asbestos problems that it has balked on its Chapter 11 commitments: "According to creditors interviewed by eBearing, their frustration with Federal-Mogul has been mounting; the company has asked for and received three extensions to the exclusivity period filing deadline. Yet the creditors told eBearing F-M was not working on a reorganization plan but instead hoped to solve its problems by lobbying Congress to restrict asbestos-related claims."
(eBearing News, "Federal Mogul Reaches Reorganization Agreement with Creditors," 2/3/03. See: http://www.eBearing.com/news2003/020301.htm)

As is the case with most firms that have gone through the special form of bankruptcy approved for asbestos liabilities, Federal-Mogul describes its Chapter 11 filing not as a prelude to lay-offs, pension fund reductions or other "going out of business" actions the term "bankruptcy" often invokes, but as a means to make the company "stronger" and "more competitive" than before, and discusses the company's commitment to growth, even while in bankruptcy:

"Our financial restructuring proceedings will permit us to continue to serve our customers while we develop a plan of reorganization that will resolve the company's asbestos liability and create a capital structure that will provide sufficient cash to fund operations and fuel new growth initiatives. This action provides a means for effectively separating our company's acquired asbestos liabilities from our true operating potential, thus paving the way for Federal-Mogul to emerge from the reorganization process as a stronger, more competitive enterprise.

5. Is Federal-Mogul going out of business?

Absolutely not. We are addressing the company's asbestos liability through the financial restructuring proceedings to preserve and strengthen our business, so that we can compete successfully in the future.

Federal-Mogul will continue to serve customers, renew current contracts, secure new business and invest in new business expansions to support our customers."
(See: http://www.federal-mogul.com/cda/content/front/0,2194,2336__4270,00.html)

Indeed, Federal-Mogul recently appeared sufficiently healthy in 2004 that it sought to acquire even more asbestos liabilities while in bankruptcy. In a widely criticized and controversial move, Federal-Mogul sought to acquire Bendix from Honeywell:

"Termed 'breathtaking in its audacity' by some and 'inspired' by others, the proposal was that F-M would acquire Bendix from Honeywell for free, taking the business and its associated asbestos liability insurance. Honeywell would finally be rid of Bendix, a division it had long tried to divest, and Federal-Mogul would gain another related product line to bolster its post-bankruptcy business operations."
(eBearing News, "Federal-Mogul and Honeywell End Bendix Acquisition Talks," Jan. 20, 2004. See: http://www.ebearing.com/news2004/012001.htm)

Though the Honeywell/Bendix deal fell through in January 2004, Federal-Mogul has reportedly continued to make investments while in bankruptcy as part of its goal of emerging "asbestos-free and with a strong balance sheet."
(eBearing News, "Federal-Mogul Reorganization Hits Setbacks," October 7, 2003. See: http://www.eBearing.com/news2003/100701.htm)

The company also claims to be meeting all the needs of their customers while in bankruptcy.

According to a review of press statements by Federal-Mogul, most of the company's job cuts over the last five years have been the result of automation and duplicative positions eliminated following the flurry of acquisitions. Other times, the company has simply blamed a tough economy for job cuts. Neither Federal-Mogul's asbestos liabilities nor its bankruptcy have ever been blamed for the job cuts in any company public relations that we are aware of.

Federal-Mogul has also remained on the Fortune 500 list, holding onto the prestige of being one of the nation's biggest companies in the country based on annual sales. Despite its bankruptcy, it holds a higher rank on the list today than it did in 1998 when it joined the list following its many acquisitions.

Federal-Mogul Fortune 500 rankings 1997-2003

Year	Rank	Revenue
1997	(Ranked #670 on Fortune 1000)	
1998	#349	$4.469 Billion
1999	#264	$6.487 Billion
2000	#298	$6.013 Billion
2001	#321	$5.457 Billion
2002	#310	$5.422 Billion
2003	#328	$5.546 Billion
2004	#328	
2005	#326	6.184 Billion

(Source: Fortune Magazine)